SENSE OF AGENCY, EMPATHY AND SCHIZOPHRENIA

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ABSTRACT

Empathy, the capacity to share the feelings of other people, is a highly important concept in the field of clinical research. It includes emotional and social- cognitive domains such as the sense of agency, emotion recognition, theory of mind and social causal attribution. Many studies show that patients suffering from schizophrenia display serious impairments in all domains. The impairments in empathic processes found in studies are predictive of patients' deficits in real world social behaviour and outcome. It is therefore important to investigate them in more detail.

(1) To investigate the relationship between two subcomponents of empathy; the sense of agency and ToM, with respect to a hierarchic association which has been hypothesized (first study). The aim was to more closely understand if empathy presents a homogeneous concept and to what extent the different subcomponents are related to each other.

(2) To study patients with ego disturbances (ED) in more detail, and to investigate whether disruptions in self- awareness are associated with social- cognitive deficits (second study).

The results from our first study show that sense of agency and ToM are dissociated processes. These results are presented in the context of recent studies suggesting that empathic processes represent an interconnected network of different, dissociable subcomponents.

The results from our second study show that ego disturbances are predicted by deficits in causal attribution, metacognition and the amount of hospitalizations. An influential model of ED assumes that the symptoms arise due to a failure in motor feedback processes. Our results point to the fact that ED arise from a broader range of impairments in the social, cognitive and clinical domain.

In the last chapter, the results of the first and the second study are discussed in more detail and conclusions regarding theoretical and methodological issues are presented.

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CHAPTER I
INTRODUCTION: EMPATHY AND SCHIZOPHRENIA

1. Definition of Empathy

Empathy is a complex construct that has been intensely studied in different research areas since a long time (de Vignemont & Singer 2006). It has been investigated for more than hundred years, and contributions have been made from very different fields of science such as developmental psychology, clinical psychology, philosophy, theology and neuroscience (Preston & de Waal 2002). The origin of the concept stems from the german word *"Einfühlung"* and was translated into English at the beginning of the 20th century. It meant a process where the observer projects himself into the object he perceives (Preston & de Waal 2002). De Vignemont & Singer (2006) point out that in today's research as many definitions of empathy are used as research groups are studying the subject. Most definitions agree on a definition stating that empathy is "an affective response more appropriate to another's situation than one's own" (Hofmann 2000) or "as an understanding of another person's feelings" (Preston & de Waal 2002). In applying such broad definitions, different phenomena can be subsumed under the term "empathy". More narrow definitions such as the one used by de Vignemont & Singer (2006) define certain conditions that must be fulfilled to distinguish empathy from similar processes such as emotional contagion or sympathy". In their definition, the term empathy is justified if: (1) the person experiences an affective state, (2) the emotional state resembles that of the object, (3) the state is elicited by the observation of the other person's state, (4) the subject understand the other person as the source of his/her own emotional state (de Vignemont & Singer 2006).

In general, empathy encompasses different aspects such as motoric, emotional and cognitive components (Preston & de Waal 2002). De Waal (2007) points out that empathic capabilities are built upon each other. He uses the analogy of a Russian doll to clarify that higher- level abilities such as ToM are founded on hard- wired basal mechanisms (de Waal 2007; Preston & de Waal 2002).

Penn et al. (2008) describes certain features that characterize empathy research. It investigates processes that take place during social contacts such as conversations or shared activities. Also, the research field is characterized by a high degree of interdisciplinary; many disciplines such as neurosciences, social psychology, neurobiology, developmental psychology and clinical psychology are involved. The goal of the research is to advice real- world applications. An example for a real- world applications is the development of specified treatment programs in the area of psychopathology such as mentalization-based approaches for borderline personality disorder (Fonagy 2000) or social- cognitive training programs for patients suffering from schizophrenia (Horan et al. 2011; Kurzban et al. 2010; Roberts et al. 2010). Another focus of research is to explore and to describe the evolutionary background of empathic processes in order to better understand their disturbance in disorders such as schizophrenia.

2. Evolutionary Background of Empathy: The Social Brain Hypothesis and Schizophrenia

In behavioural sciences, it is agreed on that primate social systems are complex entities, and that specific cognitive capacities are needed to build up and maintain social bonds (Dunbar 2009; Dunbar & Shultz 2007). Cosmides & Tooby (1989) explain that the human brain encompasses specific neural circuits, probably build to solve adaptation problems arising from the need to engage in a social environment.

Similarly, Burns (2004) argues that one of the main reasons for the evolution of the brain might have been the need to manage and move in a highly social environment. In his view, the most important problems humans had to find solutions for were of social nature, i.e. had to do with navigating the social world, meaning that probably only those neural circuits could evolve and develop that were necessary for handling social problems (Burns 2004; Preston & de Waal 2002). Dunbar (2007) assumes that the evolution of the human brain was mainly driven by the growing size of human social networks (Dunbar & Shultz 2007; Pérez-Barbería et al. 2007). In his now famous study he could show a robust correlation between the group size of primate social systems and the relative neo- cortex volume, especially with the frontal lobule volume (Dunbar & Shultz 2007; Pérez-Barbería et al. 2007). One possible interpretation assumes that social cognition, especially higher- level abilities such as ToM, need a relatively high "computational demand" (Dunbar & Shultz 2007). More specifically, the correlation seems to encompass certain social skills such as male mating strategies, the amount of social plays and tactical deception, and, as the most important one, the level of intentionality that primates are capable of. In this context, intentionality is understood as knowledge about someone else, referred to in terms such as believing or intending. Dunbar (2007) assumes that the level of intentionality primates are capable of might be a function of brain volume.

Several researchers suggest that the ability for empathic responses that differentiates humans from most animals except other primates (e.g. apes) was initiated approximately 40 - 150000 years ago by an increase in brain size and in cortical connectivity (Baron-Cohen et al. 1997; Preston & de Waal 2002). Further studies indicate that empathic processes are related to a complex reorganization of the brain, especially in intra- hemispheric connectivity in the fronto- temporal and fronto- parietal regions (Adolphs 1999; Burns 2004).

Summing up, the social brain hypothesis suggests that the demands of social life in human societies caused significant brain changes, i.e. a re- organization of the brain with interconnected neural circuits, which enable humans to successfully involve in social interaction. Burns (2004) argues that empathic abilities constitute the main difference between humans and animals, giving us a specific capacity for interpersonal relationships and culture.

In this context, disorders within the schizophrenic spectrum are understood by some researchers as an illness where the main characteristic is not the presence of positive symptoms such as delusions or hallucinations, but a profound disturbance of empathic abilities (Frith & Done 1988). In fact, Burns (2004) even describes it as "the ultimate social brain disorder". Individuals with schizophrenia normally have great difficulties to correctly read and answer social signals and often feel uncomfortable in social situations.

Results from neuroimaging studies show that the lack of empathy is accompanied by significant structural abnormalities in the area of the prefrontal cortex and the temporo- parietal junction (TPJ) (2006). The impairment of empathic abilities in schizophrenia has been shown to negatively influence the building and formation of interpersonal relationships and the social functioning in the community (Van Hooren et al. 2008).

3. Subdomains of Empathy

Empathy is a construct that encompasses a variety of subdomains (de Waal 2007). In general, an important distinction must be made between hard- wired basal mechanisms such as motor- and emotional empathy and higher- level abilities such as ToM and attribution processes.

Empathy research is focused on the following four domains: motor empathy (resonance), emotional empathy, social cognition, i.e. ToM and causal attribution processes, and the sense of agency. A brief definition of these four domains is provided in the following paragraphs.

3.1. Motor Empathy /Resonance

Blair (2005) defines motor empathy as "the tendency to automatically mimic and synchronize facial expressions, vocalizations, postures and movements with those of another person". This mimicking or mirroring is founded in the "Mirror neuron system" (MNS). These are cells in the premotor cortex that are activated when a movement is initiated, but are also active when the same movement is observed in another person (Di Pellegrino 1992). Preston & de Waal (2002) use the expression "perception-action mechanism" by which the observer has access to the subjective state of another person (the object) trough neural and body representations. De Waal (2007) pointed out that the sharing of subjective states of another person on a somatic level is an automatic process that we are sometimes unaware of. Studies using electromyography show invisible muscle contractions in the study subject's faces when watching pictures of facial expressions of other people (Dimberg et al. 2000). De Waal (2007) mentioned that the idea of shared representations between perception and action is not new and was already suggested by Lipps (1903). However, the neuroanatomical mechanism was originally discovered in 1992 in the premotor cortex of the macaque brain (Di Pellegrino 1992).

The neurons discharge when the monkey performs an action, but also when it observes someone else performing that action (Iacoboni 2009). Studies using several experimental techniques have demonstrated the existence of a MNS in the human brain (Iacoboni 2009). When an action is observed, activation can be shown in the premotor and parietal areas of the brain. The MNS is somatotopically organized. Distinct regions in the premotor and parietal areas are activated related to different actions (Gallese 2007).

It was first assumed that the neuron system functions as a kind of action recognition. But further studies discovered that the brain contains "strictly congruent mirror neurons" that fire when the same

action is executed or observed, and "broad mirror neurons" (the much larger part), which also fire when movements have the same goal or are related to each other (Iacoboni 2009). Thus, it was suggested that the function of the MNS could be the understanding of an action, and of the intention of an action (de Waal 2007). It constitutes an internal representation of different body states and its associated emotions and actions (Iacoboni 1999). Deficits in the MNS lead to difficulties in understanding other people.

3.2. Emotional Empathy

Emotional empathy refers to the experience, expression and recognition of emotions (Edwards et al. 2002; Kohler & Martin 2006; Morris et al. 2009). It is viewed as a hallmark condition for successful interaction with others and for being able to actively participate in the social environment (Kohler & Martin 2006; Kohler et al. 2010).

Six basic emotions with corresponding facial and prosodic cues are cross- culturally recognized: sadness, fear, anger, disgust, happiness and surprise. By contrast, more complex social emotions like shame, pride or flirtatiousness are dependent on very specific culture- based rules and codes (Eibl-Eibesfeldt 1970; Ekman & Friesen 1971).

In the last decades, emotion processing has become the subject of increased clinical attention and scientific interest. Research mainly focuses on exploring emotion processing in different psychiatric illnesses like depression, bipolar disorder or schizophrenia. In schizophrenia, many studies show that all aspects of experiencing, recognizing and expressing emotions are similarly affected (i.e. disturbed). Deficits in the identification of emotional facial expressions in schizophrenia were found in many studies (Edwards et al. 2002; Morris et al. 2009). It has been shown that impaired face processing is related to symptom severity and a poor functional outcome in schizophrenia patients (Brekke et al. 2005; Heimberg et al. 1992). Studies also show that emotion- processing deficits are specific for schizophrenia and not due to general performance problems (Morris et al. 2009). The deficits seem to lie in very early stages of perception (Morrison et al. 1988). Visual scan path studies have demonstrated that patients specifically tend to avoid salient regions of the face like eyes and mouth (Gordon et al. 1992; Loughland et al. 2002; Williams et al. 1999). Other studies indicate that patients prefer to stand further away from emotional faces in comparison to healthy controls. Taken together, these findings implicate that patients find emotional stimuli in general frightening and try to avoid paying particular attention (Morris et al. 2009).

Regarding underlying neurobiological structures, neuroimaging studies show that emotions are mainly processed in the right hemisphere of the brain and activate a network of different regions such as the amygdala, hippocampus, fusiform gyrus, medial and inferior frontal cortex and thalamic region (Morris et al. 2009). Some of these structures, especially the amygdala and prefrontal structures and the connections between them, are known to undergo important changes during adolescence (Perlman et al. 2004). An influential hypothesis states that emotional processing deficits in schizophrenia

patients could be due to a failure of adolescent maturation, especially of the prefrontal cortex and its connections with the amygdala (Morris et al. 2009). However, intervention studies testing the use of attention- shaping programs show that the deficits in schizophrenia can be partly remediated (Combs et al. 2008). Most research is focused on the recognition of facial cues. However, some studies investigate the prosodic recognition of emotions in schizophrenia (Edwards et al. 2002). These few studies lend support to the hypothesis that patients with schizophrenia show impairments in both processes, the recognition of prosodic and facial emotional cues (Leentjens et al. 1998).

3.3. Cognitive Empathy (Social Cognition): Theory of Mind

ToM (or mentalizing) refers to the capacity to recognize what other people intend, know or feel (Frith & Corcoran 1996; Frith & Frith 1999; Frith & Frith 2003), or, as expressed in another definition, the ability to ascribe mental states to other people (Baron-Cohen et al. 1997). ToM encompasses the understanding of other human beings as intentional agents, acting to fulfill their desires and goals. Developmental studies show that this capacity emerges between 3- 4 years of age and further develops until 11 years of age (Frith & Frith 1999). In general, philosophers discuss ToM in the context of *"intentionality"* (Dunbar 2007). ToM is associated with second order intentionality: *"I assume that you would like to ..."*. Having knowledge of one's own state of mind is described as first order intentionality (Dunbar & Shultz 2007).

In general, the growing ability to develop a ToM is understood as a very important transformation in child development. Dunbar (2007) points out that, equipped with a ToM, the child is able to perform two new activities: fictional playing and tactical deception (i.e. lying). In general, intentionality can continue up to a very high level: *"I assume that you understand that I want you to believe that..."*. The limit seems to be fifth order intentionality as a border of what humans are capable of keeping track of. Abu-Akel (2003) and others (Rochat & Striano 2002) describe abilities like goal- detection, the sense of agency, shared attention, imitation and introspection as precursor abilities or *"protoforms"* of ToM.

Developmental psychology mainly employs two sets of theories to explain the cognitive and neural processes underlying ToM. The so- called *"theory- theory"* assumes that humans learn to form a ToM by applying concepts or theories, or, differently put, by rules and axioms (Abu-Akel 2003). By contrast, the *"simulation- theory"* assumes that humans understand the other person's point of view by mental simulation of the other person's situation, by using one of their own experiences as a model. Newer models assume that both components are involved in ToM processes (Mitchell et al. 2009). ToM deficits in schizophrenia have been demonstrated in a range of studies. Deficits have been found using different tasks; in the attribution of mental states, in detecting deception and recognizing false beliefs (Corcoran et al. 1995; Pickup & Frith 2001). Meta analyses show that ToM abilities of patients are more than one standard deviation below that of healthy controls (Sprong et al. 2007).

Different hypotheses were put forward regarding the etiology of these deficits. Neuroimaging studies suggest that ToM deficits are related to disturbances in the MNS (Fahim et al. 2004). Frith (1988,

1992, 1989) proposed an important hypothesis. He suggested that certain symptoms of schizophrenia such as persecutory delusions should be understood as a result of disturbed underlying psychological processes. In his view, the inability to understand another person's intentions or beliefs (disturbed ToM) might lead to the development of delusional symptoms, feelings of being watched or spied on, and in general feelings of not being able to trust others with the extreme form of delusional symptoms. Several studies show, in line with this hypothesis, an association between disturbed ToM and paranoid symptoms (Corcoran et al. 1995). In contrast to Frith's theory, Abu-Akel (2003) states that paranoid symptoms do not indicate an absence of ToM skills, but that they might arise due to an over- attribution of knowledge to others, something he calls *"Hyper- ToM"*. In his idea, the patients do have ToM abilities, but they apply them in an atypical, unusual way.

An important issue concerns the time point when the deficits actually take place. It was suggested that schizophrenia patients have normally developed ToM skills, and that the deficits only appear after the onset of the illness in late adolescence or early adulthood (Abu-Akel 2003). By contrast, other studies indicate that ToM deficits are present before the onset of the illness and might be important for the early detection and prediction of the onset of illness (Sprong et al. 2007). Regarding the duration of deficits, investigations in patients in the acute as well as the remitted phase of the illness suggest that the deficits might be persistent (Bora et al. 2009). What more, it has been shown that the severity of deficits in the first episodes of the illness and in chronic patients are comparable, indicating that probably no further progression occurs after the onset of schizophrenia.

3.4. Cognitive Empathy (Social Cognition): Causal Attribution

Gilbert (1995) observes that people in general care more about why other people do things than about what they actually do. Explaining why and how people ascribe causes to certain events is a research field known as social causal attribution. Many different attribution models have been proposed (Heider 1958; Jones 1972; Kelley 1967).

Gilbert (1995) notes that in spite of differences in the assumptions and formulations of these theories, one common factor is that they all differentiate between external factors and internal factors lying inside a person that cause his/her behavior.

Heider (1958), the first psychologist investigating this topic, proposed exactly two factors of causation (person attribution versus environmental attribution). Later theories expanded his basic assumption.

Kelly (1967) proposed that behavior is explained by including additional information about factors co- varying with the observed behavior (factors that are present when the behavior occurs and absent when the behavior is absent). He proposed that three ways of interpretations are possible: person attribution (additional consensus information is used by checking if most people would act in a similar way), object attribution (additional consistency information is used to check if the person would generally act often or rarely in a similar way) and situational attribution (distinctiveness

information is used to analyze if the person would act similarly or differently to different stimuli). Kelley's theory is classified as an important and fundamental attribution theory, which triggered a lot of research (Harvey 2010). However, one important point of criticism is that it assumes people to logically attribute behavior in the social world as they would attribute causes of events in the physical world, not paying attention to other factors like emotions or cognitive biases (Gilbert 1995). Another attribution theory, accounting for these additional factors was proposed by Weiner (1994). He analyzed the characteristics of attribution judgments in general, concluding that attributions can be stable or instable (category of stability), internal or external (category of the locus of control) and controllable or uncontrollable (category of control). Weiner's concept has been applied in the study of psychiatric populations by searching for fundamental attribution biases correlating with different disorders. For example, many studies found that depressed people tend to attribute negative social events to internal causes, i.e. displaying a self- blaming attribution pattern (Bentall et al. 2008). Regarding schizophrenia, attribution biases have been shown in relation to delusional symptoms (Bentall et al. 1991; Bentall et al. 2008). Bentall et al. (2001) assumed that patients displaying delusional symptoms mainly attribute negative events to external factors in order to protect themselves from feelings of low self-esteem (Bentall et al. 1991). However, research only partly supports this hypothesis (Garety 1999).

3.5. Sense of Agency

The categorization of self- concepts has a tradition in philosophy and was for example worked out in the 20th century by William James who distinguished different senses of self like the physical self, the mental self, the spiritual self, and the ego (Gallagher 2000). In psychology and cognitive sciences, self- models have also been established (James 1950 ; Parnas 2005; Scharfetter 1981, 1995).

More recent approaches from philosophy and cognitive sciences differentiate mainly two aspects of self: the minimal self and the narrative self (Gallagher 2000). The minimal self (or core self) means an immediate consciousness of oneself as a subject, un- extended in time. According to Gallaher (2000) this self- experience mainly emerges from brain processes and body sensations without the subject necessarily being aware of them. The narrative self (or the extended self), by contrast, encompasses a more or less coherent self- image including a past (in form of memories) and a future (in form of wishes and intentions) (Gallagher 2000).

The sense of agency (or source monitoring) is regarded as an aspect of the minimal self. It is defined as "the sense that I am the one who is causing an action" (Gallagher 2000). Actions that are self-generated can be distinguished from externally generated actions. This gives rise to the experience of self- other distinction and thus contributes to the subjective phenomenon of self- awareness (Vogeley & Fink 2003). Several mechanisms have been proposed to explain the sense of agency by applying theories of motor- learning and control (van Holst & Mittelstaedt 1950; Wolpert & Kawato 1998). Frith &

Corcoran (1996) assume that the feeling of generating an action arises mainly from a comparison of

two sources of information; the predicted outcome (the forward model) and the actual outcome of an action. Congruence of the two sources of information indicates that oneself is the agent of the action, whereas incongruence would indicate another agent as responsible. Several studies confirm the importance of the forward model for the sense of agency (Jeannerod 2009).

However, several critical points regarding this model have been formulated. For example, Synofzik et al. (2008) or De Vignemont & Fourneret (2004) suggest that we should take into account several other factors in explaining the experience of agency, like the context of an action, or higher- level cognitive processes modulating the top- down experience of agency (Jeannerod 2009; Synofzik et al. 2008; Waters & Badcock 2008).

Experimentally, the sense of agency has been studied with movement manipulation paradigms. Nielsen (1963) created the first manipulation paradigm, an archetype for almost all future paradigms. He manipulated the visual feedback of performed movements in an experiment with healthy subjects. He showed that, despite presenting the subjects with a manipulated visual feedback, they still experienced these alien movements as their own. Nielsen (1963) interpreted his finding as an indication for the dominance of visual over kinesthetic feedback regarding the judgment of authorship in healthy subjects. Later on, Nielsen's findings stimulated the generation of important hypotheses concerning the formation of psychotic symptoms in schizophrenia (Frith 1988; Frith & Done 1989).

One hypothesis, represented by several researchers (Daprati et al. 1997; Farrer et al. 2004; Franck et al. 2001) was that in schizophrenia a disturbed sense of agency could be involved in the emergence of symptoms such as ego disturbances (ED). This idea was first formulated by Frith (1988, 1989) and was investigated in a lot of studies. The results were twofold. While several studies showed sense of agency impairments in schizophrenia patients especially in relation to ED (Daprati et al. 1997; Kircher et al. 2007; Lindner et al. 2005; Malenka et al. 1986), other studies were unable to replicate these results (Fourneret et al. 2001; Kopp & Rist 1994; Trillenberg et al. 1998).

4. The Sense of Agency and the Construct of Empathy

Many studies have shown that in situations evoking empathic responses, common neural circuits are activated for the subject and the object (Decety & Lamm 2006). The sense of agency is an important key mechanism in understanding how a subject can differentiate between his/her own feelings and that of another person. Decety & Lamm (2006) point out that disentanglement between self and other is a necessary precondition in order to adequately attribute mental states to another person. As such, the capability to distinguish between self and other allows to regulate one's emotion and to react to the situation of the other person, hence to show an empathic response (Decety & Lamm 2006). As such, the sense of agency can be understood as a precondition to the experience of empathy. This idea is backed up by developmental and clinical research.

Developmental psychology shows that the sense of agency or self- awareness develops around two years of age (Rochat & Striano 2002). Rochat & Striano (2002) suggest that the recognition of a child

in a mirror can be understood as a hallmark step in the ability to differentiate between self and other. Higher- level abilities such as ToM develop only later, around 3- 4 years of age. Developmental psychologists suggest that the emergence of ToM is only possible because of precursor abilities such as executive functions (self- regulation, planning, cognitive flexibility) and the sense of agency (Decety & Lamm 2006).

Clinical data show that in patients with schizophrenia both abilities, the sense of agency and ToM, are impaired (Daprati et al. 1997; Sprong et al. 2007). One hypothesis is that the disturbance of the sense of agency makes it more difficult for patients to understand the emotional situation of another person. Studies investigating self- rated empathic abilities in schizophrenia show that patients experience more personal distress when confronted with emotions, compared to healthy subjects. This could be one clue suggesting that dysfunctional sense of agency and dysfunctional ToM are related to each other. To further explore this hypothesis was the content of our first study.

5. Importance, Limitations and Challenges of Empathy Research in Schizophrenia

Since the uprising of the concept of empathy, it has become a highly important and intensely studied domain, especially in the field of schizophrenia research (Green et al. 2008) with an ever- growing number of investigations devoted to the above- described topics. The high amount of research has lead to the development and investigation of highly useful concepts such as ToM or social causal attribution. It has been shown that impairments in empathic functions are predictive of deficits in social outcome and real world social behavior in schizophrenia, i.e. in the ability of the patients to navigate the social world (Harvey 2010).

However, while some topics have been intensely studied, other, similarly, important research questions have not yet been addressed so far. The following questions were especially relevant for the planning of our investigation.

The first question concerns the relationship between the above- described domains of empathy. In the past, empathy was mostly studied as a one- dimensional construct, and studies used only a single measure of ToM or emotion processing. However, broadly defined it consists of several abilities (as depicted above). Therefore, it has become an important question to more clearly define which domains have to be included in the concept, and, secondly, how the particular domains are related to each other, i.e. whether they are independent or related processes, similarly to the knowledge we have about cognitive processes such as attention or concentration, and their relation to each other.

This differentiation turns out to be particularly difficult, since the different categories of empathy show a considerable overlap among each other, as it is the case in the two domains of emotion processing and ToM. Some researchers even define emotion processing as one component of ToM while others understand them as separated constructs. Related to this discussion is the question whether basic components developing very early in ontogenesis can be understood as necessary

precursor abilities. For example, it has been suggested that the sense of agency is a precursor of ToM (Decety & Chaminade 2003). Correspondingly one might ask whether deficits in ToM can be explained by dysfunctional lower level abilities in schizophrenia.

6. Project Description and Study Focus

The primary aim of our study was to investigate different aspects of empathy in a sample of schizophrenia patients and in a sample of healthy controls.

In contrast to earlier investigations and as an extension of those, we planned to use three types of measures in one study. First, we assessed the level of social functioning and symptomatology by applying the "Social Dysfunction Index" (SDI) (Munroe-Blum et al. 1996), as well as the "Positive and Negative Syndrome Scale" (PANSS) in the patient sample (Kay et al. 1987). To specifically assess ED, we used the Ego-Disturbance Scale of the "Arbeitsgemeinschaft für Methodik und Dokumentation in der Psychiatrie" (AMDP) instrument.

Secondly, we used four different measures of empathy to measure the sense of agency, emotion processing, ToM and social causal attribution.

Third, in order to evaluate the cognitive functioning of all participants, we applied measures of executive functions, verbal intelligence, concentration and attention, cognitive flexibility, word fluency and working memory. The study was conducted at the University Hospital Zurich. The tests were presented orally, on paper or directly on the computer. The duration of the experiment was approximately three hours. All participants signed an informed consent and were handed out detailed information about the study in written form.

7. Overview of the Chapters

The following chapter (chapter II) presents the results of our study on the topic of a possible relationship between the components; sense of agency and ToM. The aim was to provide research data to critically discuss the hypothesis of the sense of agency as a precursor ability of ToM that has been raised by several researchers (de Vignemont & Singer 2006; Decety & Lamm 2006). The study results, which put this hypothesis into question, are critically discussed and put in the perspective of other recent investigation data.

The third chapter presents the results of our research on the influence of one particular kind of schizophrenia symptoms, namely ED, on social- cognitive abilities. We then present our hypothesis of a specific deficit in one component of social- cognition, namely internally oriented processes such as the sense of agency and social causal attribution, following a classification of Lieberman (2007). We describe our study results and discuss them in the context of the state of research on this topic.

The final chapter provides an overview of our main results. The results are critically discussed and their empirical and practical implications are outlined, especially with regard to future studies intending to investigate social cognition in patients suffering from schizophrenia.

In our research papers, we used the term social cognition instead of empathy. In our conception, social cognition encompasses mainly the higher- level cognitive abilities such as ToM or causal attribution. Empathy is a broader construct also encompassing emotional processing, emotional contagion etc. The terms mentalizing and ToM are used synonymously.

CHAPTER II
SENSE OF AGENCY AND MENTALIZING: A DISSOCIATION OF SUBDOMAINS OF SOCIAL COGNITION IN PATIENTS WITH SCHIZOPHRENIA

1. Introduction

Social cognition is defined as the "perception, cognitive processing and interpretation of social information" (Penn 1997). Many psychiatrists regard disturbed social cognition as a main feature of schizophrenia, resulting in impaired social skills and social alienation (Brüne & Brüne-Cohrs 2006; Penn 1997). Two core processes of social cognition or more general: of empathic abilities have been intensely studied in patients with schizophrenia: "mentalizing" (also referred to as "theory of mind") and "sense of agency" (Gallagher 2000).

Sense of agency or "self- other distinction" is defined as the sense or identification of oneself as the agent of one's own actions. It enables the distinction between actions generated by oneself from those generated by others, and determines the feeling of being the author of an action (David et al. 2008) Experiments on the sense of agency are primarily philosophically or clinically motivated. Patients with schizophrenia may exhibit a specific group of symptoms, so- called first- rank symptoms, which include passivity symptoms such as thought insertion or delusions of control (Blakemore et al. 2002). Patients with these symptoms experience others' actions as a consequence of their own intentions or attribute their own actions to external sources. Several authors have explained such delusions as a breakdown of the sense of agency (Blakemore et al. 2002; Frith & Done 1988). However, schizophrenia patients without first- rank symptoms also show sense of agency impairments, albeit to a lesser degree (Daprati et al. 1997; Franck et al. 2001).

The ability to attribute mental states to oneself and other people, mentalizing (or "theory of mind"), allows a person to understand, manipulate and predict behavior (Frith & Corcoran 1996). Schizophrenia patients show significant and stable mentalizing impairments as demonstrated in numerous studies (Bora et al. 2009; Harrington et al. 2005; Sprong et al. 2007).

Mentalizing is commonly thought to be a complex social- cognitive process that depends on the development of several precursor mechanisms (Frith & Frith 1999; Stone & Gerrans 2006). Amongst other social- cognitive processes such as joint attention, gaze monitoring and the detection of animacy or intentional movements, the sense of agency has also been considered an important precursor to mentalizing (Abu-Akel 2003; Bischof-Köhler 1994; Decety & Chaminade 2003) necessary to avoid confusion about one's own and another person's beliefs or feelings. More specifically, when someone demonstrates deficits in understanding another person's false beliefs, - before understanding that this person's belief is false - he first has to recognize and evaluate his own belief and understand that the other person may have a belief different from his own (Flavell 1999; Flavell 1977). This idea is

supported by evidence from clinical populations in which both processes are subject to impairments, such as in schizophrenia. Moreover, in self- rating questionnaires, schizophrenia patients indicate lower levels of perspective taking compared to controls, but higher levels of personal distress when confronted with emotions of other people (Derntl et al. 2009; Fujiwara et al. 2008; Haker & Rössler 2009; Shamay-Tsoory et al. 2007). This could be explained by impairments in the self- other distinction, and hence a disturbed sense of agency, which could hinder the appropriate understanding of another individual's perspective. This leads us to ask: are deficits in mentalizing a mere epi- phenomenon of an inability to differentiate or disengage from other people's mental states? What is the exact nature of the relationship between sense of agency and mentalizing?

Although the self- other distinction seems fundamental to mentalizing, there is evidence suggesting that they represent two independent processes. Van Hooren et al. (2008) investigated the association between a range of social- cognitive processes, finding a lack of overlap between a sense of agency and mentalizing, and more broadly, between all the social- cognitive processes investigated. More specifically, David et al. (2008) assessed the two processes in individuals with high- functioning autism and Asperger's Syndrome (HFA/AS), which is characterized by prominent deficits in social cognition and interaction. They showed a dissociation between impaired mentalizing and an intact sense of agency in autism. In addition, evidence from functional imaging studies also suggests neurobiological dissociation or independence: David et al. (2006) for example reported that sense of agency and mentalizing, despite being assessed with the same stimulus material, showed no interaction at the neural level. In conclusion, it remains unclear whether these two processes represent two independent domains of social cognition or whether they can be conceptualized as interrelated processes. In order to further elucidate the exact nature of the relationship between the self- other distinction ability and mentalizing, and to reconcile results on dissociation between the two processes, we sought to study them in conjunction in both healthy and psychiatrically ill participants in whom impairments in both sense of agency and mentalizing processes have been shown. More specifically, we first assessed whether or not a relation between both processes could be detected by applying a widely used mentalizing test ("Reading the mind in the eyes test") and an established agency manipulation task (David et al. 2007) in a sample of schizophrenia patients and a healthy control group. Deficits in both tasks or correlated task performance in the clinical population would strongly indicate a close relationship between sense of agency and mentalizing. Second, we aimed at assessing the influence of cognitive variables and levels of social functioning on sense of agency and mentalizing by using a comprehensive neuropsychological battery and a social functioning questionnaire (SDI), in order to reveal other variables suggesting a common functional basis.

2. Method

2.1. Participants

We recruited 40 patients (28 men, 12 women) who fulfilled the ICD- 10 criteria for a diagnosis of schizophrenia and were in full or partial remission (ICD- 10 F20.x4/x5) from the outpatient service of the Psychiatric University Hospital Zurich (N = 35) and an inpatient ward of a Teaching Hospital of the University of Zurich, the "Sanatorium Kilchberg" (N = 5). Of these 40 patients, 17 were diagnosed with schizophrenia of the paranoid type, 2 of the hebephrenic type and 21 of the undifferentiated type. Symptoms were rated on the day of testing by two trained psychologists using the German version of the Positive and Negative Syndrome Scale (PANSS) (Kay et al. 1987). A total of 33 out of 40 patients received antipsychotic medicine: mean chlorpromazine equivalents (CPZe) = 410.8 (SD = 360.3); CPZe for second generation antipsychotics were calculated following Woods (2003). We assessed the presence of first- rank symptoms with the German version of the "Ego-Disturbance Scale" from the "Working Group on Methodology and Documentation in Psychiatry" instrument (AMDP) (Fähndrich & Renfordt 1985; Stieglitz & Fähndrich 1988) containing the items: thought broadcasting, thought withdrawal, thought insertion, derealization, depersonalization, other symptoms of alien control. Each item was scored from 0- 4, for a max. Score of 24 in total. In addition, 39 healthy control participants (22 men, 17 women) were recruited on the University of Zurich campus (N = 15), in adult education facilities (N = 19) and among hospital staff (N = 5).

Exclusion criteria for both groups included history of a traumatic brain injury or a neurological illness, the presence of a developmental disorder, cognitive impairment, habitual alcohol or drug abuse, vision reduction, lack of fluency in the German language and age less than 20 or over 55 years. Control participants with a history of psychiatric illness were excluded. Handedness (Edinburgh Handedness Inventory) (Oldfield 1971) and familiarity with computers (i.e., the number of hours per day spent at a computer) were assessed for each subject. Written informed consent was obtained from all participants. The study was approved by the local ethical committee and conducted in accordance with the guidelines of the Helsinki Declaration.

3. Materials and Procedure

3.1. The Agency Task

The present task can be considered an established agency manipulation task closely based on previous agency paradigms (Fourneret et al. 2001; Franck et al. 2001). Participants were seated in front of a computer screen to perform simple right- handed mouse movements towards a white object (i.e. an asterisk), which appeared either on the upper left or right side of the black screen. The movements were visible to the participants in form of a cursor. The cursor movement or visual feedback matched the participants' executed movement only in 50 % of the trials (real feedback: RF). In the other 50 % a randomly selected movement from a previous trial of the subject was replayed (false feedback: FF); for more details see David et al. (2008). The cover story was that on several trials they would not see

their own movements, but the experimenters' movements who performed the task in the next room. False movements could differ in onset, pace and path deviations. After each trial, the words *"self"* and *"other"* appeared on the screen. Participants were told to indicate whether they were the agent of the observed movement (self) or not (other) by pressing the corresponding button. Thus, a 2 x 2 design with the first factor feedback (real vs. false feedback) and the second factor evaluation (self vs. other) was employed. The task was administered on a DELL Optiplex 755 computer (1680 x 1050 resolution), using Presentation software (Neurobehavioral Systems, Albany, CA, USA). Prior to the experiment, participants practiced the task to get used to the trial procedure. The paradigm consisted of 200 trials, of which 100 were RF and 100 were FF (50 trials to the left and 50 to the right target). The task was conducted in three short runs of approximately eight minutes.

After the experiment, participants completed a debriefing questionnaire in which they had to indicate (1) the cues (temporal, spatial or both) they had used to differentiate real and false feedback, (2) how fast they had detected the false feedback and (3) how difficult they found the task.

3.2. The Mentalizing Task

Mentalizing ability was assessed with the "Reading the mind in the eyes test" (Baron-Cohen et al. 1997), which is aimed at assessing first- order mentalizing abilities. The test consists of 36 pictures showing expressive pairs of male or female eyes. Each picture is presented with four choices of adjectives. Participants are asked to choose the adjective that best describes the mental state of the person (i.e. as expressed by his/her eyes). A global accuracy Score according to Baron-Cohen et al. (2001) ranging from 0 to 36 was computed.

3.3. Neuropsychological Assessment

All participants completed a neuropsychological battery, which tapped into executive functions (Reitan 1955) (TMT- A/B), verbal intelligence (Multiple Choice Vocabulary Test) (Lehrl 2005), concentration and attention (d2) (Brickenkamp 1994), selective attention and cognitive flexibility (Victoria Stroop Test) (Regard 1981), word fluency (Aschenbrenner 2000) and working memory (digit span, Wechsler Intelligence Scale for Adults) (Wechsler 1997).

3.4. The Social Dysfunction Index (SDI)

The SDI (Munroe-Blum et al. 1996) is a questionnaire consisting of nine scales to assess social functioning with respect to nine aspects: public self, independent living, occupational functioning, family relationships, important relationships other than family, community leisure recreation, acceptance and adherence to health regimens, communication, locus of control. The overall dysfunction is calculated as a percentage of total possible Score.

4. Statistical Analysis

All statistical analyses were performed using the software SPSS 16 for Windows. Level of significance was set at $p < 0.05$ for two- tailed testing. Normal distribution of the data was verified by the Kolmogorov- Smirnov test. To investigate significant differences between patient and control group, a one- way ANOVA was applied to dependent variables, given that the normality assumption was met; group differences on non- normally distributed data were analyzed using the non- parametric Mann-Whitney test.

The performance on the sense of agency task was analyzed by computing an accuracy Score - the sensitivity index - by subtracting the number of the correct judgments from the number of incorrect judgments. The sensitivity index reflects the participants' ability to differentiate congruent movements from incongruent movements. The reaction times on self- other judgments were also computed to compare the speed with which participants decided whether a movement was judged as *"self"* or *"other"*. Additional dependent variables were the motion onset times of the movement. To investigate the potential relationship between sense of agency and mentalizing, Spearman correlation analysis was performed. The influence of socio- demographic and clinical variables on both the sense of agency and mentalizing performance was computed using Spearman correlation.

5. Results

5.1. Demographic and Clinical Sample Characteristics

The demographic and clinical characteristics of the 79 eligible participants are shown in Table 1. The group of schizophrenia participants contained a significantly higher proportion of males (67.5%) compared to the control group (55 %) ($Chi2 = 5.0$, $p < 0.05$). The schizophrenic participants were also significantly less well educated than the control group ($F = 8.7$, $p < 0.05$). There were no significant differences between groups concerning age, handedness or familiarity with computers. With respect to psychopathology, 10 participants with a Sub- Score > 3 (mean Score = 7.1 ± 1.9) on the Ego- Disturbance Scale were classified as having first- rank symptoms. This patient group showed a significantly higher PANSS Positive Score (mean Score = 18.0 ± 3.1) than patients without first- rank symptoms (mean Score = 14.6 ± 2.1) ($F = 8.1$, $p < 0.01$).

5.2. Results on Experimental Task

Participants with schizophrenia performed worse on the agency- task compared to healthy control participants. They made significantly more mistakes in judging the visual feedback correctly as self- or other- generated, as indicated by the Sensitivity Index ($F = 4.9$, $p < 0.05$) (Table 2). A closer look at the number of errors made per condition between patient and control group revealed that schizophrenia patients more often identified false feedback as self- generated ($U = 471$, $p = 0.68$, n.s.) and significantly less often as other- generated ($U = 444$, $p < 0.05$). Patients with and without first- rank symptoms did not differ significantly with respect to any of these variables. A comparison of the

rating times for the self- other judgment revealed that the patient group took significantly longer for the ratings (all $U < 431$, all $p < 0.05$). They also started significantly later with the cursor movement, compared with the control group (all $U < 309.5$, all $p < 0.05$) (see Table 2). In a debriefing questionnaire participants indicated that they recognized false feedback through temporal and spatial path deviations. Of the control participants, 15.5 % reported recognizing false feedback trials by temporal deviations (compared with 22.5 % of the patient group), while 37.5 % had used spatial deviations (35 % of the patient group) and another 47 % had used both cues (42.5 % of the patient group). There were no significant differences with regards to how fast participants (1 = very early to 5 = very late) (mean Score = 3.2 ± 1.0) and control participants (mean Score = 3.0 ± 1.0) recognized false- feedback, and how difficult they found the task (1 = very easy to 5 = very difficult) (schizophrenia patients: 2,7 ± 1.0; controls: 2.7 ± 0.9).

5.3. Results on Mentalizing Task and Correlation with Agency

In the "Reading the mind in the eyes test", the patient group identified significantly fewer mental states correctly than did healthy controls ($F = 13.1$, $p < 0.05$) (Table 2). Correlations between sense of agency and mentalizing were tested using Spearman correlation for each group as well as the different diagnostic subgroups. No significant correlations between the dependent variables of the agency and mentalizing tasks were found (all $r < 0.02$ all $p > 0.05$).

5.4. Results of the Neuropsychological Assessment

All results are displayed in Table 2. Schizophrenia patients performed significantly worse on measures of executive functioning (TMT- A/B) and verbal intelligence (MWTB), as well as on measures of concentration and attention (d2). They also showed a significantly poorer performance on measures of selective attention and cognitive flexibility (Stroop Test), working memory (digit span) and word fluency (verbal fluency test). The two groups did not differ significantly in verbal IQ, as indicated by MWTB- Scores.

5.5. The Influence of Neuropsychological, Demographic, Social Dysfunction and Clinical Variables on Sense of Agency and Mentalizing

In the schizophrenia group, the accuracy on the agency task was not influenced by any neuropsychological, demographic or social dysfunction variables. Speed of movement, (i.e. movement onset times) were positively correlated with executive functions (i.e. performance on the TMT test), age and level of social functioning. TMT- A was positively correlated with three motion onset conditions (all $r > .39$, all $p < 0.05$), whereas TMT- B was positively correlated with two motion onset conditions (all $r > .33$, all $p < 0.05$, see Table 3). Concerning age, older participants started later with cursor movement in the RS ($r = .34$, $p < 0.05$) and FS condition ($r = .33$, $p < 0.05$).

Motion onset times were also positively correlated with the following SDI subscales: occupational functioning, important relationships other than family, community functioning, leisure recreation and overall SDI Score (Table 3).

No significant influences of neuropsychological performance or demographic variables were found in the control group (all $r < 0.02$, all $p > 0.05$).

Clinical variables, especially medication (CPZE), had no influence on test performance regarding speed and accuracy in the agency and mentalizing tasks (all $r < 0.02$, all $p > 0.05$).

Neuropsychological, demographic, social functioning and clinical variables had no significant influence on mentalizing.

6. Discussion

We investigated two core processes of social cognition, namely the sense of agency and mentalizing, as well as their possible interrelationship in schizophrenia. Our results demonstrated an impairment of both processes, but no significant relation between mentalizing deficits and abnormalities in the sense of agency. The patient group also showed significant impairments in all neuropsychological tests and lower levels of social functioning. Neither neuropsychological and social functioning variables nor age had any influence on performance accuracy in the mentalizing and sense of agency tasks, but did influence the speed of movements in the agency task.

6.1. Sense of Agency Deficits in Schizophrenia

In the agency paradigm schizophrenia patients made significantly more mistakes in correctly identifying the movements than the control group (as indicated by the sensitivity index). This result was independent of executive functions, attention, concentration, working memory and cognitive flexibility. Although it is clear that explicit agency judgments naturally imply some degree of cognitive effort (i.e. "was it me or you?"), our data nevertheless indicate that sense of agency impairments constitute an additional deficit, which is present in schizophrenia patients with as well as without first- rank symptoms.

Interestingly, the patient group was not affected in identifying their own movements as self- generated. All errors with respect to the awareness and attribution of agency were committed in one direction; that is, the schizophrenia patients attributed non- congruent visual feedback as self- generated (i.e. over-attribution). Clinical observations reveal that schizophrenia is associated with both over- attributions (other- to- self direction) as well as under-attributions (self- to- other direction). This is also reflected in the schizophrenia literature with respect to speech attribution (Johns et al. 2006; Johns et al. 2001) and also with respect to incorrect agency attribution either to external sources or to the self (Daprati et al. 1997; Fourneret et al. 2001; Franck et al. 2001). In the majority of agency manipulation studies schizophrenia patients showed a tendency similar to that in our study, namely, to attribute what they see to their own agency, despite visual discrepancies (Daprati et al. 1997; Fourneret et al. 2001;

Franck et al. 2001; Synofzik et al. 2008). In this regard, we could not detect any difference in attribution style between schizophrenia patients with- and without first- rank symptoms. To date, the mechanisms accounting for the direction of attribution style remain to be revealed. It is possible that cognitive factors (Rössler & Lackus 1986), recent life experiences (Bentall et al. 1991; Blackwood et al. 2001) and paradigm characteristics (Van den Bos & Jeannerod 2002) also play a role, in addition to the specific symptoms of schizophrenia.

In contrast to previous studies, we did not detect differences in agency attribution between subjects with- and without first rank symptoms (Daprati et al. 1997; Franck et al. 2001). Several factors could account for this lack of effect: First, our patients were only mildly symptomatic at the time of testing, with possibly only minor agency attribution deficits (Spence et al. 1997). Second, we cannot rule out that our paradigm was not sensitive enough to detect subtle or very specific differences between the two patient groups. Specific impairments in relation to first- rank symptoms were mostly found in tasks employing three or more experimental conditions. For example, Franck et al. (2001) use three conditions, employing either no, angular- spatial or temporal biases between movements and feedback. They showed that patients with first- rank symptoms showed specific impairments only in the angular bias condition. Unfortunately, we employed dynamic simultaneous spatial and temporal deviations, thus not permitting specific fine- grained conclusions.

Schizophrenia patients were also impaired in other aspects of the task, namely in reaction times for judgments of agency, and also in motion onset times (i.e. they were slower in initiating the movement). Motion onset times were negatively correlated with TMT- A/B (as a measure of executive functioning, specifically psycho- motoric speed and visual attention), age and level of social functioning. These results point to a well-known motor speed deficit in schizophrenia. The deficit was more pronounced in older participants with lower executive functions and a lower level of social functioning, especially in the domains of work, social relationships, community functioning and leisure recreation (Fourneret et al. 2001; Kopp & Rist 1994).

6.2. Mentalizing Deficits in Schizophrenia

In our study, all schizophrenia patients showed significant impairments in first- order mentalizing processes. Similar results have been reported previously (Bora et al. 2009; Sprong et al. 2007). Since their first description as hallmark features in schizophrenia (Frith 1992), mentalizing deficits can be found in the acute and remitted phase of the disorder (Inoue et al. 2006; Mo et al. 2008) in first episode psychosis (Bertrand et al. 2007), schizotypy (Pickup 2006) and in relatives of schizophrenia patients (Janssen et al. 2003).

6.3. The Relationship between Mentalizing and the Sense of Agency in Schizophrenia

Our main objective was the examination of the relationship between the sense of agency and mentalizing in schizophrenia. Although the schizophrenia patients had significant deficits in both these were not related, suggesting that they represent two distinct areas of disturbance.

Previously, it had been hypothesized that the sense of agency is an important precursor for mentalizing processes, crucial for the avoidance of emotional distress and the confusion of feelings of self and other. This hypothesis has been supported so far only by indirect evidence from simultaneous impairments of both processes in clinical disorders such as schizophrenia (Blakemore et al. 2002). However, only few studies have directly investigated the relation between these two social-cognitive processes. Van Hooren et al. (2008), in a factor analysis, found no overlap between these processes among other social- cognitive processes. David et al. (2006) investigated self- other distinction and mentalizing in a functional imaging study, but found no interaction of the two processes on a neural level. More readily comparable to our own study, David et al. (2008) investigated sense of agency and mentalizing processes in HFA/AS. They found a dissociation between both processes. A direct comparison of the two studies reveals that individuals with autism (David et al. 2008) are faster in psychomotor speed in the TMT-A compared to the present patients with schizophrenia, but showed stronger mentalizing deficits in the Eyes test compared to the schizophrenia patients. With respect to agency, HFA/AS were not impaired in the attribution of movements as self or other (sensitivity index) but needed a longer time for agency judgments (as indexed by longer rating reaction times). However, differences in psychopathology and the methodological design make it difficult to draw conclusions from their study. In fact HFA/AS and schizophrenia are clinically distinct disorders that show only little overlap in their symptoms and underlying deficits. For example, in autism, mentalizing processes seem to be diminished or absent, probably because of the onset of HFA/AS in early infancy. By contrast, schizophrenia is a disorder that has its onset in mid- to late adolescence, and mentalizing processes are relatively unimpaired until the onset of the illness. After the onset, in general, mentalizing is atypical or excessive (hypermentalizing) rather than absent. Similarly, sense of agency processes are not impaired in the same way in both disorders (Abu-Akel & Bailey 2000; Frith & Done 1989), with an intact sense of agency in HFA/ AS (David et al. 2008; Russell & Hill 2001; Sebanz et al. 2005) and a disturbed sense of agency in schizophrenia (Frith & Done 1989; Knoblich et al. 2004; Malenka et al. 1986). Moreover the task requirements differed slightly (e.g., one vs. two target objects) and different experimental settings were used (not the same location, different PC and joystick/ mouse). In addition, the samples differed in age, gender, and IQ (David et al. 2008).

An alternative explanation for our finding of a dissociation might be that mentalizing encompasses a social- cognitive and a social- perceptual component, and the sense of agency might be an important precursor for the social- cognitive component in particular (Tager-Flusberg & Sullivan 2000). Neuroimaging data show that mentalizing is related to increasing activity in the prefrontal cortex (especially the medial aspect thereof), superior temporal regions and (to some extent) the amygdala (Abu-Akel 2003; Brüne & Brüne-Cohrs 2006; Frith & Frith 2003). Prefrontal regions are mainly related to the social- cognitive aspect of mentalizing, while the amygdala und superior temporal cortex are linked to the social- perceptual aspect (Tager-Flusberg & Sullivan 2000). However, it can be

assumed that the eyes test used in our study is a measure of both the perceptual and the cognitive component of mentalizing through the attribution of complex mental states (Baron-Cohen et al. 1997; Baron-Cohen et al. 2001; Shur et al. 2008). In line with this claim, two neuroimaging studies have shown the activation of temporal regions, prefrontal cortex, medial frontal lobe and the amygdala during test performance, confirming that both aspects of mentalizing are involved (Baron-Cohen et al. 1997; Russell & Hill 2001). By contrast, in schizophrenia, a hypo- activation in several brain regions, especially in the left inferior frontal gyrus (Russell et al. 2000) and right posterior orbital cortex (Brunet et al. 2003) during the performance of the eyes- test has been shown. Regarding the sense of agency, an abnormal hyperactivation of the IPL, the brain area critical for distinguishing internally produced actions from those generated by others (Decety et al. 2002; Decety & Lamm 2006; Farrer et al. 2004; Schnell et al. 2007) has been found in schizophrenia, especially in patients with first- rank symptoms (Farrer et al. 2004; Spence et al. 1997). Regarding the interaction between the sense of agency and mentalizing, it has been speculated that, since schizophrenia patients show impairments in both processes on a physiological level, the functional connectivity between the IPL and prefrontal areas, may also be impaired (Frith et al. 2000). This could perhaps explain why we could not detect a relationship between the two processes on a behavioral level. Neuroimaging studies that systematically investigate the interaction of social- cognitive processes in schizophrenia are needed to address this specific question.

6.4. Limitations

First, the patient sample was slightly biased, possibly not allowing for population inferences, as it consisted of a majority of male individuals. The average duration of illness was 15.4 years. However, this can be considered a sample composition typical for schizophrenia, since more male patients participate in clinical studies than female patients. Secondly, schizophrenia patients were less educated (in years) compared to the control participants, but this difference is not likely to explain differences on, for example, the agency task, especially because there were no significant correlations between education level and sense of agency variables. Third, it is possible that the setup of the sense of agency task used in our study may have been too abstract and as such was not clearly associated with mentalizing processes. It has been emphasized that the sense of agency is especially relevant in emotionally stressful situations i.e. where the subject interacts with another person and has to distinguish self- and other feelings in order to react properly. In our sense of agency task, the participants interacted with a computer, and no evocation of emotional distress was involved. An experiment which investigates both aspects of social cognition in a more naturalistic setup involving interaction with another agent might be needed to gain more insight into the relationship between the sense of agency and mentalizing (Davis 1996). Support for this idea can be found in studies of self-rated mentalizing abilities in schizophrenia (Derntl et al. 2009; Haker & Rössler 2009; Montag et al. 2007), where patients indicate low levels of mentalizing but high levels of personal distress compared to healthy control persons.

This indicates a relationship between self- other distinction and mentalizing. However, our agency task was based on previously used paradigms (e.g. also manipulating visual feedback on a computer screen) and can be considered a reliable indicator of sense of agency processes (Farrer et al. 2004; Fourneret et al. 2001; Franck et al. 2001) It could even be traced back to the seminal work conducted by Nielsen (1963), who had subjects draw a line on a piece of paper. They could either see their own hand tracing the line or, unbeknownst to them, the experimenter's hand, whose movements spatially deviated from the subject's own movement. That is, when the line drawn by the experimenter veered to one side (similar to our task), Nielsen's subjects adjusted their own line drawing, suggesting they were not aware of the manipulation. Additionally, our task has been previously applied in combination with fMRI and TMS in healthy participants and in a patient group with HFA/AS, demonstrating the recruitment of brain areas relevant to the sense of agency, such as the IPL (David et al. 2006; David et al. 2007).

7. Conclusion

Our findings can be interpreted as indicating a dissociation of sense of agency and mentalizing in schizophrenia. Social cognition in general is a complex construct that involves several low- and high-level processes and multiple interacting structures (Adolphs 2001; Van Hooren et al. 2008). Our findings suggest that sense of agency and mentalizing represent two non- overlapping subdomains, which independently contribute to the multifaceted construct of social cognition in schizophrenia.

A similar version of this chapter is published in "Psychiatry Research" (Schimansky et al., 2010)

Variable	Schizophrenia patient group (N = 40) M ± SD (MD)	Healthy control group (N = 39) M ± SD (MD)
Sex (Male / Female)[a]	28 /12	22 / 17
Age (Years)	38.5 ± 9.8	34.4 ± 9.7
Education (Years)[b]	12.5 ± 1.7	13.9 ± 2.1
EDI Score for Handedness[c]	87.5 ± 40.5 (100)	88.10 ± 37.3 (100)
Duration of Illness	15.4 ± 19.8	
Number of Hospitalizations	3.3 ± 4.3	
Age of Onset (years)	26.53 ± 6.5	
PANSS Positive Score	15.8 ± 3.3	
PANSS Negative Score	22.3 ± 7.2	
PANSS General Score	29.3 ± 5.1	
Hours at the Computer per Day	2.2 ± 2.0	3.0 ± 1.8

Table 1. Clinical and sociodemographic sample characteristics, PANSS Positive and Negative Syndrom (PANSS) Scores, M = mean, SD = standard deviation, MD = median [a] $Chi^2 = 5.0$, $p < 0.05$ [b] ANOVA, $F = 8.7$, $p < 0.05$ [c] Raw Score, not standardized

Dependent Variables of the Agency Task, Results on Neuropsychological Tests an Mentalizing Task	**Schizophrenia Patient Group (N = 40) M ± SD (MD)**	**Healthy Control Group (N = 39) M SD (MD)**	**Statistics**	**p**
Sensitivity Index (%)	73,0 ± 6,7	79.8 ± 10.4	F = 4,9	(p< 0.05)
Motion Onset RS	658.1 ± 182.8 (618.3)	546.2 ± 129.4 (53	U = 460	(p< 0.01)
Motion Onset RO	673.7 ± 277.3 (605.8)	537.9 ± 135.1 (55	U = 309.5	(p< 0.05)
Motion Onset FS	618.2 ± 227.9 (554.7)	496.7 ± 99.0 (468	U = 597	(p< 0.05)
Motion Onset FO	640.4 ± 192.4 (605.9)	525.9 ± 121.8 (51	U = 416	(p< 0.01)
RT on RS Judgments	1211.4 ± 816.2 (1051)	718.8 ± 268.0 (647.5)	U = 431	(p< 0.01)
RT on RO Judgments	2071.2 ± 1092.4 (1814.3)	1557.5 ± 965.3 (1543.1)	U = 337	(p< 0.05)
RT on FS Judgments	1376.9 ± 951.5 (1129.3)	1086.3 ± 620.4 (867.7)	U = 597	(p< 0.05)
RT on FO Judgments	1649.7 ± 1650.4 (1268.2)	1111.0 ± 496.0 (1039.8)	U = 454.5	(p< 0.05)
TMT- A	36.4 ± 12.1 (32)	25.58 ± 11.2 (22)	U = 346	(p< 0.01)
TMT- B	85.3 ± 31.4 (84.5)	54.2 ± 17.1 (52)	U = 262,5	(p< 0.01)
DS- V	8.7 ± 2.3 (8)	10.6 ± 2.1 (11)	U = 394	(p< 0.01)
DS- R	5.4 ± 1.5 (5)	7.4 ± 2.6 (6.4)	U = 424	(p< 0.01)
d2 (% Score)	6.2 ± 6.9 (4)	3.4 ± 3.0 (2.6)	U = 570	(p < 0.01)
Stroop Test	1.8 ± 0.4 (1.9)	1.6 ± 0.5 (1.6)	U = 478	(p < 0.05)
Fluency Test	29.9 ± 7.4 (27.5)	36.7 ± 10.4 (35)	U = 301	(p> 0.01)
MWTB- Test	28.4 ± 4.1 (29.8)	31.0 ± 0.5 (32)	U = 467,5	(p< 0.05)
IQ- Scores	108.9 ± 13.9	115.7 ± 24.9	F = 2.2	(n.s.)
Reading the mind in the eyes	23.1 ± 3.2	25.5 ± 2.6	F = 13.1	(p < 0.05)

test				

Table 2. Results of the agency task and the neuropsychological tasks for the schizophrenia group (N = 40) and the control group (N = 40) M = Mean, SD = Standard Deviation, MD = Median, RS real feedback judged as self- generated, RO real feedback judged as other-generated, FS false feedback judged as self-generated, FO false feedback judged as other-generated

	RS Onset Motion	FS Onset Motion	FO Onset Motion	R0 Onset Motion
TMT- A	0.291 p = 0.07	0.400 * p < 0.05	0.533 ** p < 0.01	0.413 * p < 0.05
TMT- B	0.291 p = 0.07	0.340 * p < 0.05	0.390 * p < 0.05	0.140 p= 0.39
SDI 3	0.376** p < 0.05	0.24** p < 0.05	0.341** p < 0.05	0.254 * p < 0.05
SDI 5	0.244 * p < 0.05	0.324 ** p < 0.05	0.274 p < 0.05	0.009 p = 0.955
SDI 6	0.045 p = 0.78	0.245* p < 0.05	0.094 p = 0.56	-0.114 p = 0.56
SDI 9	0.253* p < 0. 05	0.326 ** p < 0.05	0.249 * p < 0.05	-0.064 p = 0.69

Table 3. Correlation Matrix between neuropsychological variables and SDI scales (Social Dysfunction Index) with the Agency Variables in the Schizophrenia Patient Group (N = 40); (RS) Real Self Onset Motion, (FS) False Self Onset Motion, (FO) False Other Onset Motion, (RO) Real Other Onset Motion condition, (SDI 3 = occupational functioning, SDI 5 = important relationships other than family, SDI 6 = leisure recreation, SDI 9 = locus of control)

CHAPTER III
THE INFLUENCE OF EGO DISTURBANCES ON SOCIAL COGNITION IN PATIENTS WITH SCHIZOPHRENIA

1. Introduction

Throughout the last century, schizophrenia was traditionally referred to as an ego disorder by many psychiatrists (Bleuler 1983; Conrad 1959; Schneider 1959). This concept has been carried forward into modern psychopathology by defining individual symptoms of ED that occur in some patients. The most influential definition was provided by Schneider (1959, 1974) who termed them "first rank symptoms" because they were regarded as primary for the diagnosis of schizophrenia, including symptoms such as delusional and sense deception phenomena, thought- insertion, - withdrawal, - broadcasting and other forms of thought interference, feelings of being controlled by an external force and everything in the spheres of feelings, drive and volition which the patient experiences as imposed on him or influenced by others (Schneider 1974). Concerning a common ground of these symptoms, Schneider (1959, 1974) suggested grouping them under the concept of "permeability of the ego- world boundary", i.e. a disturbance of the ability to maintain a clear distinction between the self and the environment.

Regarding this permeability of the self, it can be assumed that patients with ego disturbances (ED) are impaired in the domain of social cognition, the ability to build representations about oneself and others as well as about relations between oneself and others (Adolphs 2001; Van Hooren et al. 2008). An intact ego- world boundary is understood as a necessary condition for an adequate understanding of the subjective world of other people (Decety & Moriguchi 2007; Forgas 2002). On the other hand, a lack of discrimination between self and other such as the perception that thoughts and actions are externally controlled or manipulated might be associated with difficulties in correctly understanding social information.

Until now, social cognition, which represents a multidimensional construct including various processes such as source monitoring, emotion recognition, mental state recognition and social causal attribution (Adolphs 1999; Van Hooren et al. 2008) has mainly been studied with regard to an influential hypothesis by Frith (Frith 1992; Frith & Done 1988, 1989). They assumed that deficits in social cognition might be selectively impaired in relation to certain symptoms of schizophrenia. In line with this hypothesis, studies using different mental state recognition paradigms such as first- and second order false belief tasks, hinting tasks and deception or animated sequence tasks, have found impairments in subjects with prominent behavioral or paranoid symptoms, but no impairments in patients with ED (Corcoran et al. 1995; Frith & Corcoran 1996; Pickup & Frith 2001; Russell et al., 2006).

Only one study reported a worse performance, which may be due to the small sample size (Corcoran et al. 1997).

By contrast source monitoring abilities have been shown to be selectively impaired in patients with ED (Daprati et al. 1997; Farrer et al. 2004; Franck et al. 2001; Knoblich et al. 2004; Lindner et al. 2005), with only a few studies showing conflicting results (Fourneret et al. 2001; Kopp & Rist 1994; Trillenberg et al. 1998).

Regarding social causal attribution, so far one study has investigated causal attribution in patients displaying ED, demonstrating a tendency to internally attribute, i.e. to blame oneself, for task- related events (Fourneret et al. 2001).

Taken together, these results could be integrated into the classification of social- cognitive processes of Lieberman and colleagues (2007; 2006) by speculating that ED might be related to impairments in internal domains of social cognition such as source monitoring or social causal attribution, which rely on reflective, mental and emotional aspects of oneself and other individuals. By contrast, processes focusing on external, physical and visual characteristics of oneself and others like mental state recognition or emotion recognition seem to be relatively intact.

However, studies that systematically investigate a range of social- cognitive processes in relation to ED are so far lacking. Therefore, the goal of our investigation was to explore this relationship in more detail in a sample of schizophrenia patients and a control sample. The specific objectives of the study were to compare the patient and the control sample on a variety of social- cognitive tasks encompassing source monitoring, emotion recognition, mental state recognition and social causal attribution, and to analyze the influence of social- cognitive and clinical variables on ED. Based on previous research we expected significant associations between source monitoring impairments, social causal attribution deficits and ED.

2. Method

2.1. Subjects: Schizophrenia Group (SG)

A group of 40 patients (28 men, 12 women, mean age = 38.3, SD = 9.8) with an ICD- 10 diagnosis of schizophrenia in full or partial remission (ICD- 10 F20.x4/x5) participated in our study. Patients were recruited from the outpatient service of the Psychiatric University Hospital Zurich (N = 35) and the inpatient ward of a Teaching Hospital of the University of Zurich, the "Sanatorium Kilchberg" (N = 5). Exclusion criteria for patients were: (1) a different main diagnosis than schizophrenia (F20.0 - F20.3), (2) age under 20 or over 60 years, (3) history of any brain injury or neurological illness, (4) vision reduction, (5) an axis II disorder (DSM- IV), (6) cognitive impairments and (7) not fluent enough in the German language to follow instructions. Of the 40 patients, 33 were receiving antipsychotic medication: mean chlorpromazine equivalents (CPZe) were 410.8 (SD = 360.3); CPZe for second-generation antipsychotics were calculated according to Woods (2003). Mean IQ, estimated by the raw Score at the Multiple Vocabulary Test B [Mehrfachwahl- Wortschatz- Intelligenztest, MWTB] (Lehrl

2005) measuring verbal- dependent intelligence was 109.0 (SD = 13.5). The average duration of illness was 15.4 years (SD = 19.8). All but two patients were right- handed (Oldfield 1971). Clinical assessment was made by personal interview on the day of testing by two trained psychologists using the German version of the "Positive and Negative Syndrome Scale" (PANSS) (Kay et al. 1987; Singh & Kay 1987). Seventeen subjects met the criteria for the paranoid type of schizophrenia, two for the hebephrenic type, and 21 for the undifferentiated type.

All patients were clinically stable at the time of testing and gave written informed consent to participate in the study, which had been approved by the local Ethics Committee. Our study was conducted in accordance with the guidelines of the Helsinki Declaration. Descriptive information for the patient and control group is provided in Table 1.

2.2. Subjects: Control Group (CG)

Thirtynine healthy control subjects (22 men, 17 women, mean age = 34.46, SD = 9.7 years) were recruited at the University of Zurich (N = 15), in adult education facilities (N = 19) and among hospital staff (N = 5). Exclusion criteria for the control subjects were: (1) history of any brain injury or neurological illness, (2) age under 20 or over 60 years (3) cognitive impairments, (4) history of habitual alcohol or drug abuse or current abuse, (5) vision reduction, (6) history of schizophrenia or related disorders; current depression (7) not sufficiently fluent in the German language to follow instructions. All but one control subject were right handed (Oldfield 1971) and the main IQ Score, estimated by the Score at the MWT- B was 115.7 (SD = 24.5) (Lehrl 2005).

3. Measures and Procedures

3.1. Ego Disturbances

We used the "Ego-Disturbance Scale" instrument from the "Working Group on Methodology and Documentation in Psychiatry" (AMDP) (Fähndrich & Renfordt 1985; Stieglitz & Fähndrich 1988) for the assessment of ED in the SG. The scale contains the following items: (1) thought broadcasting, (2) thought withdrawal, (3) thought insertion, (4) derealization, (5) depersonalization, and (6) other symptoms of alien control, following a proposal of Loftus (2000). Each item was scored from 0 - 3 (0 = non-existent, 1 = mild, 2 = medium, 3 = heavy), for a max. Score of 18 in total.

3.2. The Source Monitoring Task

Source monitoring was investigated by means of an established source monitoring task (David et al. 2007; David et al. 2008). Participants performed simple right- handed mouse movements towards a white target object (an asterisk) appearing either on the upper left or the right side of a black computer screen. Movements could be tracked visually in the form of a cursor displayed on the screen. The cursor movement matched the subjects' executed movements only in 50 % of the trials (real feedback: RF). In the other 50 % a randomly selected movement from a previous trial of the participant was

replayed (false feedback: FF). To avoid systematic differences between RF and FF, false movements always went to the same direction as the subject's intended movements, but they could differ in regard to onset, speed and path deviations. As a cover story, participants were told that on some trials they would see the experimenter's movements who performed the task in the adjacent room, instead of their own movements.

Subjects were told to monitor the movements carefully and to indicate after each trial whether they were the agent of the observed movement (self) or not by pressing the corresponding button. The task thus yielded a quasi 2x2 design with the first factor feedback (real vs. false) and the second factor evaluation (self vs. other). In a second judgment, participants indicated their confidence in their decision on a four- point rating scale (1 = certain, 2 = rather certain, 3 = rather uncertain, 4 = uncertain). (The second scale was included as an additional rating to assess the confidence with which the evaluation of self or other was made.)

The task was conducted on a DELL Optiplex 755 computer (1680 x 1050 resolution), employing the Presentation software (Neurobehavioral Systems, Albany, USA). Each participant read standardized written instruction before the test started, followed by 20 practice trials to become used to the task procedure. The task consisted of a total of 200 trials, of which 100 were RF and 100 were FF (50 trials to the left and 50 trials to the right target). It was carried out in three short runs of approximately eight minutes.

3.3. The Emotion Recognition Task

The emotion recognition task was designed by Edwards (2001) and demanded the identification of six basic emotions by means of spoken sentences: sadness, anger, happiness, disgust, surprise and fear. The content of the sentences was neutral and consisted of simple statements, for example: "She will drive fast". To test the ability of the subjects to understand general prosodic cues, a control task was used consisting of two questions and two declarative sentences that had to be correctly identified before starting the test. The main test was composed of 12 sentences. Each emotion appeared two times. Including the four control sentences, the task contained 16 trials. Response categories were coded as 0 = incorrect and 1 = correct.

3.4. The "Reading the mind in the eyes test"

The "Reading the mind in the eyes test" is aimed at assessing mental state recognition abilities (Baron-Cohen et al. 1997). The test consisted of 36 pictures showing expressive pairs of male or female eyes. Each picture was presented with four choices of adjectives. Subjects were asked to choose the adjective that best describes the mental state of the person (i.e. as expressed by his/her eyes). A global accuracy Score according to Baron-Cohen et al. (1997) ranging from 0 to 36 was computed.

3.5. The Attribution Task

The attribution task used in our study is based on the attribution theory of Kelley (1967) and was designed by Rössler & Lackus (1986). Kelley (1967) proposed that each action, behavior or event can be attributed to three categories of causes: the person that performs an action, the object the action is directed to or the circumstances or situation in which the action takes place. While person and object categories can be understood as relatively stable categories, attributing a cause to the circumstances of a situation is more of an unstable and transient causal category. Kelley assumed that in order to make the correct attribution, the observer analyses all available information in terms of three categories, namely consensus, distinctiveness and consistency. We use "Susie is afraid of the dog" as an example: Consensus (across persons): is high if several other people are afraid of the dog and low if nobody else is afraid of the dog. In the case of low consensus, the observer would attribute the fear to Susie´s character as a stable disposition.

Distinctiveness (over entities): is high if Susie is only afraid of this dog and low if Susie is afraid of all dogs. In the case of high distinctiveness the observer would conclude that this dog is responsible for Susie´s fear.

Consistency (over time): is high if Susie is always afraid of the dog and low if Susie is afraid of the dog only once. In the case of low consistency the observer would conclude that situational circumstances are responsible Susie´s fear.

In our setup, the participants were presented with depictions of 18 written social events on a computer screen. Each description of an event was presented three times, in each case with changing additional information regarding the consensus, distinctiveness and consistency of the event, for example: Susie is afraid of the dog. 1) Nobody else is afraid of this dog (low consensus). 2) Susie is not afraid of other dogs (high distinctiveness). 3) Usually Susie is not afraid of this dog (low consistency). Each description indicated a specific causal attribution (person, object, and situation) according to the theory of Kelley (1967). To eliminate interfering variables, none of the depictions of social events contained references to emotional states. A person attribution error was classified if participants made false person attributions in cases where demanded object or situational attributions were called for. Similarly, object and situational errors were classified if participants did not choose the appropriate attribution.

4. Statistical Analysis

Data were analyzed using SPSS version 19 software for Windows (SPSS Inc., Chicago, IL). The normal distribution of data was verified by the Kolmogorov- Smirnov test. Analyses of significant differences between the study groups were calculated by applying a one- way ANOVA, given that the normality assumption was met. Non- normally distributed variables were analyzed using the Mann-Whitney U- Test. The Chi2 test was used to analyze possible age differences between groups.

In a first step ANOVAs were used to determine whether there were significant differences in socio-demographic variables and in the social- cognitive task performance between the CG and the SG. As dependent variables for the source monitoring task we used (1) the sensitivity index (number of correct trials- number of incorrect trials) and (2) the degree of confidence of the self- other evaluation, which was calculated as a new variable; the mean confidence Score (the sum of all four ratings divided by the total number of ratings, resulting in a number between 1- 4). As dependent variables for the emotion recognition task the number of correctly identified general prosodic cues in the control task and of correctly identified emotions in the main task were used. For the mental state recognition task the number of correctly identified mental states was used as dependent variable. For the attribution test false person, object and situational attributions were used as dependent variables. Bivariate Pearson and Spearman correlations between ED and social- cognitive and clinical variables were calculated. In a second step, a stepwise regression analysis was used to model if the degree of ED in the SG could be predicted by social- cognitive functioning and by clinical variables. A positive correlation between ED and the PANSS positive scale was found, which represents an overlap between the concepts of ego disorder and positive symptoms. Therefore the PANSS positive scale was excluded as explaining variable from the regression analysis. Levels of significance were set at $p < 0.05$.

5. Results

5.1. Comparison between the SG and the CG

Descriptives of the two samples regarding sociodemographic data, clinical data and results of the social- cognitive tasks are shown in Table 1. SG and CG differed significantly in education ($U = 503.5$, $p < 0.01$) and in all social- cognitive tasks (all $F > 3.6$, all $p < 0.05$), with the SG being less educated and performing worse in all applied social- cognitive tasks. The only measure that was not different was the confidence rating in the source monitoring task, where the SG showed the same amount of confidence in their self- other evaluation as the CG ($F = 0.0$, $p > 0.05$).

5.2. Correlation Analysis between ED and Social Cognitive and Clinical Measures

To evaluate the relationship between ED and social- cognitive, sociodemographic and clinical variables, correlation analyses were performed (Table 2). From the social- cognitive variables, the ED Score was significantly correlated with the confidence rating of the source monitoring task. With respect to clinical variables, correlations were found with the number of hospitalizations and with the PANSS Positive Score.

5.3. Regression Analysis Between ED and Social Cognitive and Clinical Measures

Table 3 shows the hierarchical regression analysis for ED. The results of the analysis show that a higher number of person attribution errors ($ß = 0.61$, $p < 0.05$), elevated levels of confidence in the

self- other rating (ß = 0.51, $p < 0.01$) and a higher number of hospitalizations (ß = 0.38, $p < 0.01$) were significant predictors for the presence of ED. The three independent variables accounted for 61.8 % of the variance in ego- disturbances ($F = 16.0$, $p < 0.01$).

6. Discussion

The present study compared four domains of social cognition in a sample of schizophrenia patients and a sample of healthy controls and, in a second step, analyzed the relationship between social-cognitive and clinical variables and the presence of ED.

In line with previous research we found that schizophrenia patients had deficits in all four examined domains of social cognition (Aakre et al. 2009; Bora et al. 2009; Brüne & Juckel 2010; Kohler et al. 2010; Mizrahi et al. 2008). According to our hypothesis, the performance in the social- cognitive tests explained almost half of the variance of the ego disturbance Score. Within the social- cognitive battery, internally oriented social- cognitive domains such as social causal attribution or the metacognitive confidence judgment in the source monitoring task were predictive for ED, whereas externally-oriented processes such as basic emotion recognition or mental state recognition were not predictive variables. The number of hospitalizations also had a predictive effect. Contrary to our hypothesis, no association was found between the self- other evaluation in the source monitoring task and ED.

So far, in line with our results, a number of studies have demonstrated significant deficits in all investigated domains of social cognition with a clear impact on functional outcome and implications for treatment guidelines (Bora et al. 2009; Couture et al. 2006; Marwick & Hall 2008). Regarding the specific deficits in relation to ED, different factors have been discussed (Leube & Pauly 2008; Nasrallah 1985; Trimble 1990). It has been suggested that abnormalities such as temporal lobe pathology (Trimble 1990), abnormal activity in the parietal lobe (Danckert et al. 2004; Leube et al. 2008), alterations in associative higher- order neuronal circuits such as the mirror- neuron system (Leube & Pauly 2008; Peled et al. 2003), reduced cerebral asymmetry (Waters 2009) or time perception deficits (Waters & Jablensky 2009) might be relevant factors associated with ED. Regarding social-cognitive processes, only source monitoring deficits, important in securing self- other differentiation, have been discussed as central for the formation of ED (Blakemore et al. 2002; Leube et al. 2008; Lindner et al. 2005). Our results suggest that deficits in other internally focused social- cognitive processes like social causal attribution could be similarly important factors.

Social causal attribution in general must be understood as a process encompassing internally and externally oriented aspects of social cognition since causes of events can be attributed to persons or to external factors like objects or situations. In line with this differentiation, neuro- imaging evidence shows that person attribution is associated with activation of brain areas related to internally oriented activity (Harris et al. 2005). In a more concrete sense, an internalizing attribution style characterized by a high number of person attribution errors means that a subject is considered responsible for an

event, not an object or a situation. Rössler & Lackus (1986) point out that a preference for stable (person or object) over unstable categories (situation) can be understood as an attempt to gain control and make the outside world more predictable. Situation attributions are complex and imply little prognostic value for future events. Person attributions, by contrast, are easier und allow forecasts of the behavior of people in future situations. Our study results suggest that an inflexible attribution style with an overemphasis on person attribution might be a risk factor for the emergence of ED (Jeannerod 2009). In line with our results, a study of Fourneret (2001) using a source monitoring task shows that patients with ED were unable to attribute the differences between visual information and motor information externally to the computer, but attributed them to internal factors such as tiredness, lack of concentration or effects of the disease. Symptoms of ED in general are associated with a great loss of control over the self and self- experience. Attributing causes of events to a person might be an attempt to restore control. The negative effect of this strategy is, however, the creation of fear and isolation, since events cannot be adequately understood and judged. In line with this idea, Mizrahi et al. (2008) show that a high level of person attributions is associated with more psychopathology and less response to treatment. In this regard, it would be interesting for a future study to use attribution tasks investigating real- life attribution patterns derived from patients' narratives such as reported in a study of Aakre et al. (2009) to more closely understand the nature of attribution deficits in this patient group.

Regarding the source monitoring task, contrary to our hypothesis we did not find an association between these performance deficits and ED. Several factors could account for this negative finding. First, our source monitoring paradigm might not have been subtle and complex enough to detect specific deficits in patients with ED. In other studies, paradigms with several experimental conditions were used. For example Daprati et al. (1997) used a paradigm with three experimental conditions and found differences only in the most complex condition, where subject and experimenter had to perform exactly the same movement. By contrast, in our task the subjects could use temporal as well as spatial cues to evaluate movements as self- or other generated. Secondly, it is possible that mild ED are not related to deficits in action monitoring but to a lower confidence in one´s own performance. So far, several studies have shown that patients with schizophrenia exhibit deficits in metacognition, i.e. the judgment of one´s own performance in a test (Bacon et al. 2001; Kircher et al. 2007). However, the relationship between metacognition and ED has not yet been investigated. Morrison et al. (1997) found no relationship between symptoms of hallucinations and metacognition. Moritz et al. (2002) found a positive relationship between symptoms of delusions and hallucinations and elevated levels of confidence in a word learning task, however, passivity experiences were not included either. Reduced levels of confidence in one´s own performance have been demonstrated for other psychiatric disorders like depression (Corcoran 2008; Soderstrom et al. 2011) and obsessive- compulsive disorder (OCD) (Macdonald et al. 1997; Zitterl et al. 2001), leading to specific symptoms like rumination (depression) or checking (OCD). Regarding schizophrenia, more studies including patients with moderate to severe

symptoms of ED are needed to investigate whether ED are associated with lower confidence in all performance tasks or only in source monitoring tasks, and whether patients with stronger symptoms, by contrast, show higher levels of confidence as suggested in the study of Moritz et al. (2002).

The third significant predictor in our study is the number of hospitalizations. Previous studies have established hospitalization as a predictor for several negative outcomes such as a higher risk for suicide (Bowers et al. 2010), relapse (Ascher-Svanum et al. 2010; Doering et al. 1998), switching antipsychotics (Sernyak 2005), the experience of criticism and hostility from relatives (Bentsen et al. 1998), a more severe course of illness and lower social functioning (Vetter 1996). In our study, it was the only clinical variable predicting ED. One interpretation could be that a higher number of positive symptoms, especially symptoms of ED, involve an increased risk of being hospitalized, as research suggests (Kozma et al. 2010). However, more studies are needed to validate our findings.

Lastly, in the recognition of emotions and the perception of mental states, no specific impairments in relation to ED were detected. These findings support our hypothesis that ED per se seem not to interfere with externally oriented domains of social cognition.

Finally, some methodological points deserve consideration. First, as mentioned above, the patients with ED were only mildly symptomatic at the time of testing and encompassed only a small number of patients. Therefore, our results have to be interpreted with caution and should be replicated in larger samples.

Second, it is possible that the results are explainable by the fact that the patients with ED were more severely ill than the other patients as suggested by the correlation of the ED Score with the PANSS Positive Score and with the number of hospitalizations. However, and arguing against this explanation, we did not find a correlation of the ego- disturbance Score with any other clinical variables. The high correlation between the PANSS Positive Score and the ego- disturbance Score suggests rather that by applying the AMDP scale our concept of ED might have been too narrow. Only passivity items and two items associated with delusions (derealization, depersonalization) were included. Sense deception items like hallucinations and other delusional items like grandiosity were not included, as suggested by other instruments such as the SAPS scale or the ICD- 10 criteria. The correlation between the PANSS positive scale and the AMDP scale shows that ED should be investigated with instruments encompassing all three types of phenomena; passivity symptoms, sense deception symptoms and delusional phenomena, as described by Schneider and others (Fourneret et al. 2001; Koehler 1979; Waters & Jablensky 2009; Waters 2009). Additionally, ED were not associated with a higher PANSS negative or PANSS general Scores, suggesting that the patient group with ED was not more severely ill.

To sum up, a high number of person attributions, a lower confidence in the own performance in the source monitoring task and a higher number of hospitalizations were the main predictors for ED. The deficits were mainly found in the sphere of internally focused processes, following an important classification of Lieberman (Lieberman 2007; Satpute & Lieberman 2006). Our findings have clear

implications for clinical practice. Cognitive and behavioral interventions that specifically target patients´ attribution style and their confidence regarding their ability in self- other differentiation may help to treat the symptoms of individuals suffering from ED. Our results also suggest that the impact of this symptomatology should not be only studied in the area of neurobiology and motor cognition, but also in the area of social cognition, where heterogeneity considering specific deficits in the schizophrenia spectrum disorder has been shown.

A similar version of this chapter was published in "Psychopathology" (Schimansky et al., 2011)

	Schizophrenia Group (N = 40)			Control Group (N = 39)				
	mean	**SD**	**MD**	**mean**	**SD**	**MD**	**Statistics**	**p**
Sociodemographic variables								
Sex (Male:Female)	28:12			22:17			$X^2 = 1.5$	n.s.
Age, years	38.5	9.8		34.4	9.7		F (1,77) = 3.1	n.s.
IQ	109	13.5		115.7	24.5		F (1,77) = 2.2	n.s.
Handedness (Right:Left)[a]	87.5	40.5	100	88.10	37.3	100	U = 760	n.s.
Education (Years)	12.5	1.7	13	13.9	2.1	13	U = 503.5	<0.05
Illness- Related Variables								
Duration of Illness (Years)	15.4	19.8						
Number of Hospitalizations (Years)	3.3	4.3						
PANSS Positive	15.8	3.3						
PANSS Negative	22.3	7.2						
PANSS General	29.3	5.1						
Mean Antipsychotic Dose, CPZM	410.8	360.3						
Social- Cognitive Variables								
Source Monitoring Task								
Sensitivity Index	73	6.7		79.8	10.4		F (1,77) = 4.9	< 0.05
Certainty Rating	1.4	0.3		1.4	0.3		F (1,77) = 0.0	n.s.
Emotion Recognition in Prosody	8.1	8.5	8.5	8.9	1.4	8.5	U = 569.5	< 0.05
Reading the mind in the eyes test	23.1	3.1		25.5	2.6		F (1,77) = 12.9	< 0.01
Attribution Task								
Incorrect Person Attribution	5.7	5.4		3.2	2.7		F (1,77) = 12.8	< 0.01
Incorrect Object Attribution	9.5	4.0		7.0	3.5		F (1,77) = 8.3	< 0.05
Incorrect Circumstantial Attribution	3.6	2.6		2.4	2.0		F (1,77) = 4.2	< 0.05

Table 1. Sociodemographic variables of the Sample; MD = Mean Deviation, [1] Raw Score, not standardized

Sociodemographic Variables	**Pearson's r**	**p**
Age, Years	0.287	n.s.
IQ	-0.038	n.s.
Education (Years)	-0.027	n.s.
Illness- Related Variables		
Duration of Illness (Years)	0.020	n.s.
Number of Hospitalizations (Years)	0.348	<0.05
PANSS Positive	0.491	<0.001
PANSS Negative	0.144	n.s.
PANSS General	0.223	n.s.
Mean Antipsychotic Dose, CPZM	-0.094	n.s.
Social- Cognitive Variables		
Source Monitoring Task		
Sensitivity Index	0.208	n.s.
Certainty Rating	0.343	<0.05
Emotion Recognition in Prosody	0.091	n.s.
Reading the mind in the eyes test	0.117	n.s.
Attribution Task		
Incorrect Person Attribution	0.345	<0.05
Incorrect Object Attribution	0.260	n.s.
Incorrect Circumstantial Attribution	0.129	n.s.

Table 2. Relationship between social- cognitive and clinical variables and the ego disturbance score

	corr DR^2	F	p
Modell 1	0.171	6.764 (1,27)	<0.050
Modell 2	0.480	13.900 (2,26)	<0.000
Modell 3	0.618	16.083 (3,25)	<0.000
	b	T	p
Modell 1			
Incorrect Person Attributions	0.448	2.601	<0.05
Modell 2			
Incorrect Person Attributions	0.609	4.296	<0.000
Certainty index	0.585	4.126	<0.000
Modell 3			
Incorrect Person Attributions	0.610	5.019	<0.000
Certainty index	0.518	4.202	<0.000
Number of Hospitalizations	0.383	3.225	<0.000

Table 3. Linear regression analysis (stepwise) with the ego disturbance score as the dependent variable

CHAPTER IV
GENERAL DISCUSSION

1. Overview of the Results

In the following chapter we discuss limitations and strengths of our study project and draw some general conclusions. An overview of the major aims and results is presented in Table 1.

Chapter	Main research question	Results	Conclusions
I	To study the relationship of two main social- cognitive abilities; the sense of agency and TOM in patients with schizophrenia and healthy controls.	The results yielded a dissociation between the sense of agency and ToM.	Deficits in high- level social-cognitive abilities are not explained by baseline deficits. Our results indicate that the relationship of all components of social cognition must be studied in more detail.
II	To explore if patients with ED, a subgroup of schizophrenia patients, show deficits in social cognition compared to patients without ED.	Regression analysis showed that social- cognitive variables (attribution style, confidence in one's own performance in the agency task) and clinical variables (number of hospitalizations) explained more than half of the variance in the presence of ED.	Social- cognitive abilities are important explanatory variables for the presence of ED, in addition to neurobiological variables and aspects of motor cognition. They should be accounted for in treatment programs and in further studies of ED.

Table 1. Summary of our study's findings

2. Conclusions

2.1. The construct of empathy

Our first study, presented in Chapter II, investigated the hypothesis of an interrelationship between the sense of agency and ToM. The results of this study are an important contribution to the on- going discussion about empathy and its components.

The lack of an association between the sense of agency and ToM is in line with a host of studies demonstrating that empathic abilities are not related to each other. In this way, empathy, and also social cognition differ from the concept of neuro- cognition. Factor analyses show the existence of a neurocognitive network with highly correlated components (Van Hooren et al. 2008). These findings were replicated in studies with schizophrenia patients, suggesting a common factor underlying cognitive deficits (Dickinson et al. 2007). For empathy, the state of research is much less consistent. Green et al. (2011) pointed out that the research is hindered by the fact that terms, definitions and subdomains vary, which makes communication and a comparison of findings highly difficult. To organize and better understand the broad concept of empathy, different classification schemes have been arranged. For example, Shamay-Tsoory et al. (2007) classified the abilities into cognitive and affective aspects of cognition. Other classifications spoke about internally and externally focused abilities, or, about reflective and reflexive abilities (Lieberman 2007), or about motor empathy (resonance), emotional empathy and cognitive empathy. However, so far, the different classifications just add to the confusion, because different research groups present different definitions of the various components.

Despite these difficulties, the importance of the construct of empathy, and of its cognitive part, social cognition, for the understanding of mental disorders is undisputed. The Institute of Mental Health (NIMH) included empathy tests in the "Treatment Research to Improve Social Cognition in Schizophrenia" (MATRICS). It was argued that empathic abilities are a mediating variable between neurocognition and the functional outcome in schizophrenia (Green et al. 2011).

2.2. Suggestions for studies investigating empathy

To enhance the comparability of empathy research, future studies should employ a consistent, homogenous concept, including a standard definition of its sub- components. One possibility would be to rely on the important definition by Green et al. (2008). They include the following components: emotion processing, ToM, social perception, social knowledge and attribution bias. Employing this definition can be useful because at the NIMH workshop where the definition was built up, some of the most important researchers were participating, and the declared goal of the workshop was to address the obstacle of inconsistent terminology (Green et al. 2008).

Also, it is useful to closely study broader sub components such as social cognition, which encompasses two aspects: social causal attribution and ToM. It is important to study the relationship between neurocognition and social cognition in more detail. In this regard, we refer to an important study by Ziv et al. (2011) investigating the relation between ToM, emotion recognition and cognitive abilities. They found a close relationship between ToM tasks and tests demanding analytical reasoning abilities. Other studies such as Langdon & Coltheart (2001) or Pinkham and Penn (2008) failed to show correlations between neuro- and social cognition.

Adding neurocognitive tests to studies investigating empathy might be a different, but interesting way to better understand the neural foundation of this concept and to enlighten the discussion about the different subcomponents.

2.3. Attribution style and Ego Disturbances (ED)

The results of our second study suggest that an inflexible attribution style i.e. an overemphasis on person attribution is a risk factor for ED.

Interestingly, Bentall (2001) has formulated a hypothesis assuming that certain ED such as delusions arise as a result of abnormal attribution processes (Bentall et al. 2008; Bentall et al. 2009; Blackwood et al. 2001). Healthy individuals show a tendency to internal- personally attribute positive outcomes of events and to external- personally attribute negative events, the "Self- Serving Bias". A study by Campbell & Sedikides (1999) showed that this bias is most activated when the self of people is threatened (for example by negative outcomes of events). Studies investigating patients with paranoia found out that they have a strong tendency to attribute negative events to other persons (i.e. an exaggerated self- serving bias), neglecting situational explanations.

Bentall (2001) offered two possible explanations for this phenomenon: Either patients pay more attention to other people during negative experiences, thus fostering paranoid ideas about them; or, as a second or additional possibility, patients fail to adequately process situational information. In this regard, Bentall (2001) stressed the cognitive aspect, referring to a study of Gilbert et al. (1988), which showed that individuals under cognitive load more readily attribute negative events to other people than to situational factors.

Rössler & Lackus (1986) pointed out that the preference for stable categories might be an attempt to gain control and predictability for future situations. In addition, Bentall (2001) stressed that attributions to other persons normally make individuals feel better by avoiding self- blame. This function might be more pronounced in individuals with psychotic symptoms. The above results suggest that person attributions serve a self- protecting function and require less cognitive energy than searching for additional information as would be necessary for situational attributions.

Bentall's work is very useful for the interpretation of our findings, since he explicitly investigated the role of attribution style in the occurrence and maintenance of symptoms. However, it is difficult to apply this to our results for two reasons. First, our definition of ED, following the AMDP instrument,

did not encompass delusions. Second, our attribution paradigm was not differentiated enough to grasp the differences between internal- personal and external- personal attribution style. It had only three categories: person, object, and situation. For a specific relation to the hypothesis of Bentall it would be important to differentiate between an internal- personal attribution (oneself) and an external- personal attribution (the other person).

However, similar to our findings, Bentall (2001) also pointed out that several variables are necessary for the development of delusions. Besides the attribution style, he named factors such as a specific attention to threat- related stimuli and better emotion recognition for genuine emotions. A study investigating emotion recognition found that paranoid patients show a better recognition for genuine emotions of surprise and for negative emotions than healthy controls (Davis 2000).

Bentall (2001) also strived for complex models, denying that only one factor is responsible. Another study by Mancuso et al. (2011) also stressed the close relationship between attribution style and clinical variables. They found that from several social- cognitive factors, attribution style had the closest association with clinical variables such as positive symptoms.

Future studies should use more differentiated instruments to assess attribution style in patients with ED. In this regard, Aakre et al. (2009) carried out a very interesting study. They used samples of participant's reports about positive and negative life events, which were then coded for positivity or negativity and for internality vs. externality („The Leeds Attributional Coding System"). They were able to show that patients with current paranoid symptoms used more external- personal attributions for negative events of their own lives. We think that this is a very interesting result. It demonstrates the need to further study the relationship of ED and attribution style, but with a more specified attribution instrument that allows grasping the difference between internal and external attribution.

2.4. Critical Points: Terminology

Early psychiatric conceptualizations depict ED as a main feature of schizophrenia (Bleuler 1911). Many phenomenological oriented psychiatrists follow this description, characterizing ED as a fundamental syndrome of schizophrenia (Conrad 1959; Parnas 1982; Sass 2003; Scharfetter 1981, 1995; Schneider 1959). In this way, different definitions and understandings of ED have developed. This is for example expressed in the fact that several terms exist describing similar but not identical groups of symptoms. These are: ED, first- rank symptoms, nuclear symptoms (Loftus et al. 2000), ego pathology (Röhricht 2004), or passivity symptoms (Waters 2009).

One important difference is whether these terms are used in a phenomenological or in a diagnostic sense. In this regard "first- rank symptoms", an expression introduced by Schneider (1959), makes a diagnostic claim, defining which symptoms can be regarded as primary for the diagnosis of schizophrenia. These symptoms encompass passivity symptoms (experiences of bodily influences, thought withdrawal and other forms of thought interference), but also sense deception (the hearing of one's thought spoken aloud in one's head, the hearing of voices commenting on what one is doing at

the time, voices arguing in the third person) and delusional phenomena such as delusional perception (Koehler 1979).

Most of the other terms are mainly used in a descriptive way. However, the problem regarding the interpretation study results is that different symptoms are grouped under these terms in such a way that they are hard to compare. For example, Waters & Jablensky (2008) used the terms "passivity symptoms" and "first- rank symptoms" synonymously, but they also investigated sense deception phenomena (hallucinations) that are not included in other studies investigating passivity symptoms. Nordgaard (2008) pointed out that a lot of studies only present very unclear definitions of the symptoms investigated, or worse, no definition at all. The use of different symptom groups could be one reason for heterogeneous results. In our view, it is of utmost importance to clearly define which symptoms are included or left out.

Similarly, the terms derealization and depersonalization are defined as ego disturbance in the AMDP instrument but do not occur anywhere else. The comparability with other studies using different instruments is thus limited.

The results of our study suggest that the symptoms measured with the AMDP instrument are correlated to the presence of positive symptoms, which in our view speaks for a broad definition, including sense deception and delusional phenomena. The problem of lacking comparability has been stressed by many researchers (Koehler 1979; Nordgaard 2008; Scharfetter 1981; Trimble 1990). One attempt to resolve some of these critical issues is the development of new research tools. An example is the "Examination of Anomalous Self Experience" interview (EASE) (Parnas 2005). The EASE is an extensive interview based on the description of patients' experiences. It encompasses several domains such as: cognition and stream of consciousness, self- awareness and presence, bodily experiences, demarcation and existential reorientation. In a broader sense, it comprises not only ED, but, more generally, disorders of the self. Parnas et al. (2005) described the purpose of the checklist as „a detailed account of phenomena that have in common a somehow deformed sense of first- person perspective, in brief, a disorder or deficiency in the sense of being a subject, a self- coinciding center of action, thought and experience. In our view, this is an interesting and fruitful approach grasping the symptoms in a more encompassing and meaningful way. In general, ED are harder to classify than any other symptoms of schizophrenia. In our experience, patients often have problems to clearly decide whether they experience ED or not. Parnas et al. (2005) explained this by the fact that patients often are short of words and have difficulties explaining their own experiences because they possess „a pre- reflective quality". Additionally, they pointed out that schizophrenia diminishes the patient's ability to express themselves (negative symptoms).

However, a major disadvantage of the interview is that it is very long. The procedure takes several hours, which makes it impractical for clinical research encompassing various instruments and tests. However, it should be mentioned here as an outstanding approach to a more precise and comprehensive understanding of the nature of ED. The patients themselves as such stress the

importance of the concept. One patient, C. Kean (2009) wrote: *"Schizophrenia has silenced my real self, and even the observing self is biased by the process of subjective observation. In my opinion, schizophrenia is ultimately a disorder of the self, a disturbance of one's subjective self- experience and the external or objective reality."*

3. Outlook

We suggest that more studies investigating empathy and ED might yield important and unique contributions to the field of social cognition and psychopathology in schizophrenia.

In this sense, Waters et al. (2011) investigated if specific social impairments exist in patients with ED. They compare 227 patients with and without ED on measures of everyday social interaction (encompassing items such as conversation skills, communication by body language, self- presentation, affect display). Their findings showed that patients with ED are more severely impaired in social interactions, which leads to the conclusion that the study demonstrates an association between ED and social behavior. The authors suggested that ED are the result of an extensive deficit in body representation; not only of disturbed sensory motor processes, as Frith's model suggests (Frith 1992). In their view, ED also concern body processes and psychology and motor processes. In this regard, we hope that further studies are able to expand and add to the very important model of Frith (1992; 1988) concerning ED and disturbed underlying neurobiological processes.

Curriculum Vitae

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August 2009	„Sozialkognitive Fähigkeiten bei Schizophrenie“, Vortrag auf der klinikinternen Forschungskonferenz, Universitätsklinikum Köln
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FÖRDERUNG

2008 - 2010	Mitglied im Peer Mentoring Projekt der Universität Zürich, Organisation und Besuch von fachübergreifenden Workshops zum laufbahnfördernden Kompetenzerwerb
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SPRACHKENNTNISSE

Deutsch	Muttersprache
Englisch	Fliessend in Wort und Schrift
Französisch	Verständigung in Wort und Schrift
Indonesisch	Grundkenntnisse
Russisch	Grundkenntnisse

PUBLIKATIONEN

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Haker, H., Schimansky, J. & Rössler, W. (2010). Sociophysiology: Basic processes of empathy. *Neuropsychiatry* 2010; 24(3):151-60.

Schimansky, J., Haker, H,. & Rössler, W.(2012). The influence of social cognition on ego disturbances in patients with schizophrenia. *Psychopathology* 45 (2):117-125

Haker, H., Schimansky, J., Jann, S., & Rössler, W. (2012). Self-reported empathic abilities in schizophrenia: A longitudinal perspective. Psychiatry Research, doi:10.1016/j.psychres.2012.04.004.

BIBLIOGRAPHY

Aakre, J. M., Seghers, J. P., St-Hilaire, A., & Docherty, N. (2009). Attributional Style in Delusional Patients: A Comparison of Remitted Paranoid, Remitted Nonparanoid and Current Paranoid Patients with Nonpsychiatric Controls. *Schizophrenia Bulletin* 35(5): 994-1002.

Abu-Akel, A. (2003). A Neurobiological Mapping of Theory of Mind. *Brain Research Reviews* 43(1): 29-40.

Abu-Akel, A., & Bailey, A. L. (2000). The possibility of different forms of theory of mind impairments in psychiatric & developmental disorders. Letter. *Psychological Medicine* 30(03): 735-738.

Adolphs, R. (1999). Social Cognition and the Human Brain. *Trends in Cognitive Sciences* 3(12): 469-479.

Adolphs, R. (2001). The Neurobiology of Social Cognition. *Current Opinion in Neurobiology* 11(2): 231-239.

Aschenbrenner, S., Tucha, O., Lange, K. W. (2000). Regensburger Wortflüssigkeitstest (RWT) Göttingen: Hogrefe.

Ascher-Svanum, H., Nyhuis, A. W., Stauffer, V., Kinon, B. J., Faries, D. E., Phillips, G. A., et al. (2010). Reasons for Discontinuation and Continuation of Antipsychotics in the Teatment of Schizophrenia from Patient and Cinician Perspectives. *Current Medical Research and Opinion* 26(10): 2403-2410.

Bacon, E., Danion, J.-M., Kauffmann-Muller, F., & Bruant, A. (2001). Consciousness in Schizophrenia: A Metacognitive Approach to Semantic Memory. *Consciousness and Cognition* 10(4): 473-484.

Baron-Cohen, S., Jolliffe, T., Mortimore, C., & Robertson, M. (1997). Another Advanced Test of Theory of Mind: Evidence From Very High Functioning Adults With Autism or Asperger Syndrome. *Journal of Child Psychology and Psychiatry* 38(7): 813-822.

Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The "Reading the Mind in the Eyes" Test Revised Version: A Study with Normal Adults, and Adults with Asperger Syndrome or High-Functioning Autism. *Journal of Child Psychology and Psychiatry* 42(2): 241-251.

Bentall, R. P., Corcoran, R., Howard, R., Blackwood, N., & Kinderman, P. (2001). Persecutory Delusions. A Review and Theoretical Integration. *Clinical Psychology Review* 21(8): 1143-1192.

Bentall, R. P., Kaney, S., & Dewey, M. E. (1991). Paranoia and Social Reasoning: An Attribution Theory Analysis. *The British Journal of Clinical Psychology* 30 13-23.

Bentall, R. P., Rouse, G., Kinderman, P., Blackwood, N., Howard, R., Moore, R., et al. (2008). Paranoid Delusions in Schizophrenia Spectrum Disorders and Depression: The Transdiagnostic Role of Expectations of Negative Events and Negative Self-esteem. *The Journal of Nervous and Mental Disease* 196(5).

Bentall, R. P., Rowse, G., Shryane, N., Kinderman, P., Howard, R., Blackwood, N., et al. (2009). The Cognitive and Affective Structure of Paranoid Delusions: A Transdiagnostic Investigation of Patients With Schizophrenia Spectrum Disorders and Depression. *Archives of General Psychiatry* 66(3): 236-247.

Bentsen, H., Notland, T. H., Boye, B., Munkvold, O. G., Bjøge, H., Lersbryggen, A. B., et al. (1998). Criticism and Hostility in Relatives of Patients with Schizophrenia or Related Psychoses: Demographic and Clinical Predictors. *Acta Psychiatrica Scandinavica* 97(1): 76-85.

Bertrand, M. C., Sutton, H., Achim, A. M., Malla, A. K., & Lepage, M. (2007). Social-Cognitive Impairments in First-Episode Psychosis. *Schizophrenia Research*: 124-133.

Bischof-Köhler, D. (1994). Selbstobjektivierung und fremdbezogene Emotionen. Identifikation des eigenen Spiegelbildes, Empathie und prosoziales Verhalten im 2. Lebensjahr. *Zeitschrift für Psychologie* 202 (4), 349-377.

Blackwood, N. J., Howard, R. J., Bentall, R. P., & Murray, R. M. (2001). Cognitive NeuropsychiatricModels of Persecutory Delusions. *American Journal of Psychiatry* 158(4): 527-539.

Blair, R. J. R. (2005). Responding to the Emotions of Others: Dissociating Forms of Empathy through the Study of Typical and Psychiatric Populations. *Consciousness and Cognition* 14: 698-718.

Blakemore, S. J., Wolpert, D. M., & Frith, C. D. (2002). Abnormalities in the Awareness of Action. *Trends in Cognitive Sciences* 6(6): 237-242.

Bleuler, E. (1911). Dementia Praecox oder die Gruppe der Schizophrenien Leipzig / Wien:Deuticke.

Bleuler, E. (1983). Lehrbuch der Psychiatrie. Berlin, Heidelberg, New York: Springer.

Bora, E., Yucel, M., & Pantelis, C. (2009). Theory of Mind Impairment in Schizophrenia: Meta-Analysis. *Schizophrenia Research* 109(1-3): 1-9.

Bowers, L., Banda, T., & Nijman, H. (2010). Suicide Inside: A Systematic Review of Inpatient Suicides. *The Journal of Nervous and Mental Disease* 198(5): 315-328.

Brekke, J., Kay, D. D., Lee, K. S., & Green, M. F. (2005). Biosocial pathways to functional outcome in schizophrenia. *Schizophrenia Research* 80(2-3): 213-225.

Brickenkamp, R. (1994). Test D2 Aufmerksamkeits-Belastungstest Göttingen: Hogrefe.

Brüne, M., & Brüne-Cohrs, U. (2006). Theory of Mind-Evolution, Ontogeny, Brain Mechanisms and Psychopathology. *Neuroscience & Biobehavioral Reviews* 30(4): 437-455.

Brüne, M., & Juckel, G. (2010). Soziale Kognition bei Schizophrenien. *Der Nervenarzt* 81(3): 339-346.

Brunet, E., Sarfati, Y., Hardy-Bayle, M. C., & Decety, J. (2003). Abnormalities of Brain Function During a Nonverbal Theory of Mind Task in Schizophrenia. *Neuropsychologia.* 41(12): 1574-1582.

Burns, J.(2004). An Evolutionary Theory of Schizophrenia: CorticalConnectivity, Metarepresentation and the Social Brain. *Behavioral and Brain Sciences* 27: 831-855.

Campbell, W. K., & Sedikides, C. (1999). Self-Threat Magnifies The Self-Serving Bias: A Meta-Analytic Integration. *Review of General Psychology3*: 23-43.

Combs, D. R., Tosheva, A., Penn, D. L., Basso, M. R., Wanner, J. L., & Laib, K. (2008). Attentional-Shaping as a Means to Improve Emotion Perception Deficits in Schizophrenia. *Schizophrenia Research* 105(1-3): 68-77.

Conrad, K. (1959). Die beginnende Schizophrenie. Versuch einer Gestaltanalyse des Wahns. Stuttgart: Thieme.

Corcoran, K. M., Segal, Z.V. (2008). Metacognition in Depressive and Anxiety Disorders: Current Directions. *International Journal of Cognitive Therapy* 1(2): 33-44.

Corcoran, R., Cahill, C., & Frith, C. D. (1997). The Appreciation of Visual Jokes in People with Schizophrenia: A Study of 'Mentalizing' Ability. *Schizophrenia Research* 24(3): 319-327.

Corcoran, R., Mercer, G., & Frith, C. D. (1995). Schizophrenia, Symptomatology and Social Inference: Investigating "Theory of Mind" in People with Schizophrenia. *Schizophrenia Research* 17(1): 5-13.

Cosmides, L., Tooby, J. (1989). Evolutionary Psychology and the Generation of Culture. Part II. Case Study: A Computational Theory of Social Exchange. *Ethology and Sociobiology* 10: 51-97.

Couture, S. M., Penn, D. L., & Roberts, D. L. (2006). The Functional Significance of Social Cognition in Schizophrenia: A Review. *Schizophrenia Bulletin* 32(suppl 1): S44-S63.

Danckert, J., Saoud, M., & Maruff, P. (2004). Attention, Motor Control and Motor Imagery in Schizophrenia: implications for the Role of the Parietal Cortex. *Schizophrenia Research* 70(2-3): 241-261.

Daprati, E., Franck, N., Georgieff, N., Proust, J., Pacherie, E., Dalery, J., et al. (1997). Looking for the Agent: an Investigation Into Consciousness of Action and Self-Consciousness in Schizophrenic Patients. *Cognition* 65(1): 71-86.

David, N., Bewernick, B. H., Cohen, M. X., Newen, A., Lux, S., Fink, G. R., et al. (2006). Neural Representations of Self versus Other: Visual-Spatial Perspective Taking and Agency in a Virtual Ball-tossing Game. *Journal of Cognitive Neuroscience* 18(6): 898-910.

David, N., Cohen, M. X., Newen, A., Bewernick, B. H., Shah, N. J., Fink, G. R., et al. (2007). The Extrastriate Cortex Distinguishes Between the Consequences of one's own and others' Behavior. *NeuroImage* 36(3): 1004-1014.

David, N., Gawronski, A., Santos, N. S., Huff, W., Lehnhardt, F. G., Newen, A., et al. (2008). Dissociation Between Key Processes of Social Cognition in Autism: Impaired Mentalizing but Intact Sense of Agency. *Journal of Autism and Developmental Disorders* 38(4): 593-605.

Davis, M. H. (1996). Assessment of Individual Differences in Empathy. In J. Harvey (Ed.), *Empathy: A Social Psychological Approach* (Vol. 2 pp. 46-61). Colorado: Westview Press.

Davis, P. J., Gibson, M.G. (2000). Recgnition of Posed and Genuine Facial Expressions of Emotion in Paranoid and Nonparanoid Schizophrenia. *Journal of Abnormal Psychology* 109: 445-450.

De Vignemont, F. & Fourneret, P. (2004). The Sense of Agency: A philosophical and empirical review of the “Who” system. *Consciousness and Cognition* 13, 1-19.

De Vignemont, F., & Singer, T. (2006). The Empathic Brain: How, When and Why? *Trends in Cognitive Sciences* 10(10): 435-441.

De Waal, F. B. M. (2007). The "Russian Doll" model of Empathy and Imitation. In S. Braten (Ed.), *On Being Moved*. Amsterdam / Philadelphia: John Benjamins Publishing Company.

Decety, J., & Chaminade, T. (2003). When the Self Represents the Other: A New Cognitive Neuroscience View on Psychological Identification. *Consciousness and Cognition* 12(4): 577-596.

Decety, J., Chaminade, T., Grezes, J., & Meltzoff, A. N. (2002). A PET Exploration of the Neural Mechanisms Involved in Reciprocal Imitation. *Neuroimage.* 15(1): 265-272.

Decety, J., & Lamm, C. (2006). The Role of the Right Temporoparietal Junction in Social Interaction: How Low-Level Computational Processes Contribute to Meta-Cognition. *Neuroscientist* 13(6): 580-593.

Decety, J., & Moriguchi, Y. (2007). The Empathic Brain and its Dysfunction in Psychiatric Populations: Implications for Intervention across Different Clinical Conditions. *BioPsychoSocial Medicine* 1: 1-22.

Derntl, B., Finkelmeyer, A., Toygar, T. K., Hülsmann, A., Schneider, F., Falkenberg, D. I., et al. (2009). Generalized Deficit in all Core Components of Empathy in Schizophrenia. *Schizophrenia Research* 108(1-3): 197-206.

Di Pellegrino, G., Fadiga, L., Fogassi, L., Gallese, V., Rizzolatti, G. (1992). Understanding Motor Events: A Neurophysiological Study. *Journal of Cognitive Neuroscience* 10: 640-656.

Dickinson, D., Bellack, A. S., & Gold, J. M. (2007). Social / Communication Skills, Cognition And Vocational Functioning in Schizophrenia. *Schizophrenia Bulletin* 33: 1213-1320.

Dimberg, U., Thunberg, M., & Elmehed, K. (2000). Unconsciuos facial reactions to emotional facial reactions. *Psychologocial Science* 11: 86-89.

Doering, S., Müller, E., Köpcke, W., Pietzcher, A., Gaebel, W., Linden, M., et al. (1998). Predictors of Relapse and Rehospitalization in Schizophrenia and Schizoaffective Disorder. *Schizophrenia Bulletin* 24(1): 87-98.

Dunbar, R. I. M. (2007). Social Brain Hypothesis and Its Relevance to Social Psychology. In J. P. Forgas, M. G. Haselton & W. Von Hippel (Eds.), *Evolution and the Social Mind; Evolutionary Psychology and Social Cognition*. New York: Psychology Press.

Dunbar, R. I. M., & Shultz, S. (2007). Understanding Primate Brain Evolution. *Philosophical Transactions of the Royal Society B: Biological Sciences* 362(1480): 649-658.

Edwards, J., Jackson, H. J., & Pattison, P. E. (2002). Emotion Recognition via Facial Expression and Affective Prosody in Schizophrenia: A Methodological Review. *Clinical Psychology Review* 22(6): 789-832.

Edwards, J., Pattison, P. E., Jackson, H. J., & Wales, R. J. (2001). Facial Affect and Affective Prosody Recognition in First-Episode Schizophrenia. *Schizophrenia Research* 48(2-3): 235-253.

Eibl-Eibesfeldt, I. (1970). Ethology: The Biology of Behaviour New York: Holt, Rinehart & Winston.

Ekman, P., & Friesen, W. V. (1971). Constants Across Cultures in the Face and Emotion. *Journal of Personality and Social Psychology* 17(2): 124-129.

Fahim, C., Stip, E., Mancini, A., Boualem, M., Malaspina, D., & Beauregard, M. (2004). Negative Socio-Emotional Resonance in Schizophrenia: A Functional Magnetic Resonance Imaging Hypothesis. *Medical Hypotheses* 63(3): 467-475.

Fähndrich, E., & Renfordt, E. (1985). The AMDP System for the Documentation of Psychiatric Symptoms: Course and Effectivity of A Training Seminar. *Pharmacopsychiatry* 18(4): 278-281.

Farrer, C., Franck, N., Frith, C. D., Decety, J., Georgieff, N., d'Amato, T., et al. (2004). Neural Correlates of Action Attribution in Schizophrenia. *Psychiatry Research* 131(1): 31-44.

Flavell, J. (1999). Cognitive Development: Children's Knowledge About the Mind. *Annual Review of Psychology* 50: 21-45.

Flavell, J. H. (1977). The Development of Knowledge About Visual Perception. *Nebraska Symposium on Motivation* 25: 43-76.

Fonagy, P. (2000). Attachment and Borderline Personality Disorder. *Journal of the American Psychoanalytic Association* 48(4): 1129-1146.

Forgas, J. P., Williams, K.D. (2002). The Social Self New York: Psychology Press.

Fourneret, P., Franck, N., Slachevsky, A., & Jeannerod, M. (2001). Self-Monitoring in Schizophrenia Revisited. *Neuroreport* 12(6): 1203-1208.

Franck, N., Farrer, C., Georgieff, N., Marie-Cardine, M., Dalery, J., d'Amato, T., et al. (2001). Defective Recognition of One's Own Actions in Patients with Schizophrenia. *The American Journal of Psychiatry* 158(3): 454-459.

Frith, C. D. (1992). The Cognitive Neuropsychology of Schizophrenia Hove, UK: Lawrence Erlbaum Associates.

Frith, C. D., Blakemore, S., & Wolpert, D. M. (2000). Explaining the Symptoms of Schizophrenia: Abnormalities in the Awareness of Action. *Brain Research - Brain Research Reviews* 31(2-3): 357-363.

Frith, C. D., & Corcoran, R. (1996). Exploring 'Theory of Mind' in People with Schizophrenia. *Psychological Medicine* 26(3): 521-530.

Frith, C. D., & Done, D. J. (1988). Towards a Neuropsychology of Schizophrenia. *The British Journal of Psychiatry : The Journal of Mental Science* 153: 437-443.

Frith, C. D., & Done, D. J. (1989). Experiences of Alien Control in Schizophrenia Reflect a Disorder in the Central Monitoring of Action. *Psychological Medicine* 19(2): 359-363.

Frith, C. D., & Frith, U. (1999). Interacting Minds-A Biological Basis. *Science* 286(5445): 1692-1695.

Frith, U., & Frith, C. D. (2003). Development and Neurophysiology of Mentalizing. *Philosophical Transactions of the Royal Society of London - Series B: Biological Sciences* 358(1431): 459-473.

Fujiwara, H., Shimizu, M., Hirao, K., Miyata, J., Namiki, C., Sawamoto, N., et al. (2008). Female Specific Anterior Cingulate Abnormality and its Association with Empathic Disability in Schizophrenia. *Progress in Neuro-Psychopharmacology and Biological Psychiatry* 32(7): 1728-1734.

Gallagher, S. (2000). Philosophical Conceptions of the Self: implications for Cognitive Science. *Trends in Cognitive Sciences* 4(1): 14-21.

Gallese, V. (2007). Before and Below Theory of Mind: Embodied Simulation and the Neural Correlates of Social Cognition. *Philosophical Transactions of the Royal Society B: Biological Sciences* 362: 659-669.

Garety, P. A., Freeman, D. (1999). Cognitive Approaches to Delusions : A Critical Review of Theories and Evidence. *British Journal of Clinical Psychology* 38: 113-154.

Gilbert, D. T. (1995). Attribution and Interpersonal Perception. In A. Tesser (Ed.), *Advanced Social Psychology*. New York: McGraw-Hill.

Gilbert, D. T., Pelham, B.W., Krull, D.S. (1988). On Cognitive Busyness: When Person Perceivers Meet Persons Perceived. *Journal of Personality and Social Psychology* 54: 733-740.

Gordon, E., Coyle, S., Anderson, J., Healey, P., Cordaro, J., Latimer, C., et al. (1992). Eye Movement Response to a Facial Stimulus in Schizophrenia. *Biological Psychiatry* 31(6): 626-629.

Green, M. F., Bearden, C. E., Cannon, T. D., Fiske, A. P., Hellemann, G. S., Horan, W. P., et al. (2011). Social Cognition in Schizophrenia, Part 1: Performance Across Phase of Illness. *Schizophrenia Bulletin* 38(4): 854-864.

Green, M. F., Penn, D. L., Bentall, R., Carpenter, W. T., Gaebel, W., Gur, R. C., et al. (2008). Social Cognition in Schizophrenia: An NIMH Workshop on Definitions, Assessment, and Research Opportunities. *Schizophrenia Bulletin* 34(6): 1211-1220.

Haker, H., & Rössler, W. (2009). Empathy in Schizophrenia: Impaired Resonance. *European Archives of Psychiatry and Clinical Neuroscience* 259(6): 352-361.

Haker, H., Schimansky, J., Rössler, W. (2010). Soziophysiologie: Grundlegende Prozesse der Empathiefähigkeit. *Neuropsychiatrie* 24(3): 151-160.

Harrington, C., Siegert, R.J. & Mc Clue, J. (2005). Theory of mind in schizophrenia: a critical review. *Cognitive Neuropsychiatry* Aug 10 (4): 249-286.

Harris, L. T., Todorov, A., & Fiske, S. T. (2005). Attributions On the Brain: Neuro-Imaging Dispositional Inferences, Beyond Theory of Mind. *NeuroImage* 28(4): 763-769.

Harvey, P. D., Penn, D. (2010). Social Cognition: The Key Factor Predicting Social Outcome in People With Schizophrenia? *Psychiatry* 7(2): 41-44.

Heider, F. (1958). The Psychology of Interpersonal Relations. New York: Wiley.

Heimberg, C., Gur, R. E., Erwin, R. J., Shtasel, D. L., & Gur, R. C. (1992). Facial Emotion Discrimination: III. Behavioral Findings in Schizophrenia. *Psychiatry Research* 42(3): 253-265.

Hofmann, M. L. (2000). Empathy and Moral Development: Implications for Caring and Justice. Cambridge: University Press.

Iacoboni, M. (2009). Imitation, Empathy and Mirror Neurons. *Annual Review of Psychology* 60: 653-670.

Iacoboni, M., Woods, R.P., Brass, M., Bekkering, H., Mazziotta, J.C., Rizzolatti, G. (1999). Cortical Mechanisms of Human Imitation. *Science* 286: 2526-2528.

Inoue, Y., Yamada, K., Hirano, M., Shinohara, M., Tamaoki, T., Iguchi, H., et al. (2006). Impairment of Theory of Mind in Patients in Remission Following First Episode of Schizophrenia. *European Archives of Psychiatry and Clinical Neuroscience* 256(5): 326-328.

James, W. (1950). The Principles of Psychology. Dover Publications.

Janssen, I., Krabbendam, L., Jolles, J., & van Os, J. (2003). Alterations in Theory of Mind in Patients with Schizophrenia and Non-Psychotic Relatives. *Acta Psychiatric Scandinavia* 108(2): 110-117.

Jeannerod, M. (2009). The Sense of Agency and its Disturbances in Schizophrenia: a Reappraisal. *Experimental Brain Research* 192(3): 527-532.

Johns, L. C., Gregg, L., Allen, P., & McGuire, P. K. (2006). Impaired Verbal Self-Monitoring in Psychosis: Effects of State, Trait and Diagnosis. *Psychological Medicine.* 36(4): 465-474.

Johns, L. C., Rossell, S., Frith, C., Ahmad, F., Hemsley, D., Kuipers, E., et al. (2001). Verbal Self-Monitoring and Auditory Verbal Hallucinations in Patients with Schizophrenia. *Psychological Medicine* 31(4): 705-715.

Jones, E. E., Nisbett, R.E. (1972). The Actor and the Observer: Divergent Perceptions of the Causes of Behaviour. In E. E. Jones, Kanouse, D.E., Kelley, H.H., Nisbett, R.E., Valins, S., Weiner, B. (Ed.), *Attribution: Perceiving the Causes of Behaviour* (pp. 79-94). Morristown: General Learning Press.

Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The Positive and Negative Syndrome Scale (PANSS) for Schizophrenia. *Schizophrenia Bulletin* 13(2): 261-276.

Kean, C. (2009). Silencing the Self: Schizophrenia as a Self-Disturbance. *Schizophrenia Bulletin* 35(6): 1034-1036.

Kelley, H. H., Levine, D. (1967). Attribution Theory in Social Psychology. *Nebraska Symposium on Motivation*.

Kircher, T. T., Koch, K., Stottmeister, F., & Durst, V. (2007). Metacognition and Reflexivity in Patients with Schizophrenia. *Psychopathology* 40(4): 254-260.

Knoblich, G., Stottmeister, F., & Kircher, T. (2004). Self-Monitoring in Patients with Schizophrenia. *Psychological Medicine* 34(8): 1561-1569.

Koehler, K. (1979). First-Rank Symptoms of Schizophrenia: Questions Concerning Clinical Boundaries. *British Journal of Psychiatry* 134: 236-248.

Kohler, C. G., & Martin, E. A. (2006). Emotional Processing in Schizophrenia. *Cognitive Neuropsychiatry* 11(3): 250 - 271.

Kohler, C. G., Walker, J. B., Martin, E. A., Healey, K. M., & Moberg, P. J. (2010). Facial Emotion Perception in Schizophrenia: A Meta-analytic Review. *Schizophrenia Bulletin* 36(5): 1009-1019.

Kopp, B., & Rist, F. (1994). Error - Correcting Behavior in Schizophrenic Patients. *Schizophrenia Research* 13(1): 11-22.

Kozma, C., Dirani, R., Canuso, C., & Mao, L. (2010). Predicting Hospital Admission and Discharge with Symptom or Function Scores in Patients with Schizophrenia: Pooled Analysis of a Clinical Trial Extension. *Annals of General Psychiatry* 9(1(24)).

Langdon, R., & Coltheart, M. (2001). Visual Perspective-Taking and Schizotypy: Evidence for a Simulation-Based Account of Mentalizing in Normal Adults. *Cognition* 82: 1-26.

Leentjens, A. F., Wielaert, S. M., van Harskamp, F., & Wilmink, F. W. (1998). Disturbances of Affective Prosody in Patients with Schizophrenia; A Cross Sectional Study. *Journal of Neurology, Neurosurgery & Psychiatry.* 64(3): 375-378.

Lehrl, S. (2005). Der Mehrfach-Wortschatz Intelligenztest MWTB. Spitta Verlag.

Leube, D., & Pauly, K. (2008). Ich-Störungen-Psychologie. In T. Kircher, Gauggel, S. (Ed.), *Neuropsychologie der Schizophrenie*. Heidelberg: Springer.

Leube, D., Whitney, C., & Kircher, T. (2008). The Neural Correlates of Ego-Disturbances (Passivity Phenomena) and Formal Thought Disorder in Schizophrenia. *European Archives of Psychiatry and Clinical Neuroscience* 258 Suppl 5: 22-27.

Lieberman, M. D. (2007). Social Cognitive Neuroscience: A Review of Core Processes. *Annual Review of Psychology* 58: 259-289.

Lindner, A., Thier, P., Kircher, T. J., Haarmeier, T., & Leube, D. T. (2005). Disorders of Agency in Schizophrenia Correlate with an Inability to Compensate for the Sensory Consequences of Actions. *Current Biology* 15(12): 1119-1124.

Lipps, T. (1903). Einfühlung, Innere Nachahmung und Organempfindung. *Archiv für die gesamte Psychologie* 1: 465-519.

Loftus, J., Delisi, L. E., & Crow, T. J. (2000). Factor Structure and Familiality of First-Rank Symptoms in Sibling Pairs with Schizophrenia and Schizoaffective Disorder. *The British Journal of Psychiatry : The Journal of Mental Science* 177: 15-19.

Loughland, C. M., Williams, L. M., & Gordon, E. (2002). Schizophrenia and Affective Disorder Show Different Visual Scanning Behavior for Faces: A Trait versus State - Based Distinction? *Biological Psychiatry* 52(4): 338-348.

Macdonald, P. A., Antony, M. M., Macleod, C. M., & Richter, M. A. (1997). Memory and Confidence in Memory Judgments Among Individuals with Obsessive Compulsive Disorder and Non-Clinical Controls. *Behaviour Research and Therapy* 35(6): 497-505.

Malenka, R. C., Angel, R. W., Thiemann, S., Weitz, C. J., & Berger, P. A. (1986). Central Error-Correcting Behavior in Schizophrenia and Depression. *Biological Psychiatry* 21(3): 263-273.

Mancuso, F., Horan, W. P., Kern, R. S., & Green, M. F. (2011). Social Cognition in Psychosis: Multidimensional Structure, Clinical Correlates, and Relationship with Functional Outcome. *Schizophrenia Research* 125: 143-151.

Marwick, K., & Hall, J. (2008). Social Cognition in Schizophrenia: A Review of Face Processing. *British Medical Bulletin* 88(1): 43-58.

Mitchell, P., Currie, G., & Ziegler, F. (2009). Two Routes to Perspective: Simulation and Rule-Use as Approaches to Mentalizing. *British Journal of Developmental Psychology* 27(3): 513-543.

Mizrahi, R., Addington, J., Remington, G., & Kapur, S. (2008). Attribution Style as a Factor in Psychosis and Symptom Resolution. *Schizophrenia Research* 104(1-3): 220-227.

Mo, S., Su, Y., Chan, R. C., & Liu, J. (2008). Comprehension of Metaphor and Irony in Schizophrenia During Remission: the Role of Theory of Mind and IQ. *Psychiatry Research* 157(1-3): 21-29.

Montag, C., Heinz, A., Kunz, D., & Gallinat, J. (2007). Self-Reported Empathic Abilities in Schizophrenia. *Schizophrenia Research* 92(1-3): 85-89.

Moritz, S., & Woodward, T. S. (2002). Memory Confidence and False Memories in Schizophrenia. *The Journal of Nervous and Mental Disease* 190(9): 641-643.

Morris, R. W., Weickert, C. S., & Loughland, C. M. (2009). Emotional Face Processing in Schizophrenia. *Current Opinion in Psychiatry* 22(2): 140-146.

Morrison, A., & Haddock, G. (1997). Cognitive Factors in Source Monitoring and Auditory Hallucinations. *Psychological Medicine* 27(03): 669-679.

Morrison, R. L., Bellack, A. S., & Mueser, K. T. (1988). Deficits in Facial-Affect Recognition and Schizophrenia. *Schizophrenia Bulletin* 14(1): 67-83.

Munroe-Blum, H., Collins, E., McCleary, L., & Nuttall, S. (1996). The Social Dysfunction Index (SDI) for Patients with Schizophrenia and Related Disorders. *Schizophrenia Research* 20(1-2): 211-219.

Nasrallah, H. A. (1985). The Unintegrated Right Hemispheric Consciousness as Alien Intruder: A Possible Mechanism for Schneiderian Delusions in Schizophrenia. *Comprehensive Psychiatry* 26(3): 273-282.

Nielsen, T. (1963). Volition: A New Experimental Approach. *Scandinavian Journal of Psychology* 4: 225-230.

Nordgaard, J., Sidse, M.A., Handest, P., Parnas, J. (2008). The Diagnostic Status of First-Rank Symptoms. *Schizophrenia Bulletin* 34: 137-154.

Oldfield, R. C. (1971). The Assessment and Analysis of Handedness: The Edinburgh Inventory. *Neuropsychologia* 9(1): 97-113.

Parnas, J., Möller, P., Kircher, T., Thalbitzer, J., Jansson, L., Handest, P., Zahavi, D. (2005). EASE: Examination of Anomalous Self-Experience. *Psychopathology* 38: 236-258.

Parnas, J., Schulsinger, F., Schulsinger, H., Mednick S.A., Teasdale, M.A. (1982). Behavioral Precursors of Schizophrenia Spectrum: A Prospective Study. *Archives of General Psychiatry* 38: 658-663.

Peled, A., Pressman, A., Geva, A. B., & Modai, I. (2003). Somatosensory Evoked Potentials During a Rubber - Hand Illusion in Schizophrenia. *Schizophrenia Research* 64(2-3): 157-163.

Penn, D. L., Corrigan, P.W., Bentall, R.P., Racenstein, J.M. (1997). Social Cognition in Schizophrenia. *Psychological Bulletin* 121(1): 114-132.

Pérez-Barbería, F. J., Shultz, S., & Dunbar, R. I. M. (2007). Evidence for Coevolution of Sociality and Relative Brain Size In Three Orders of Mammals. *Evolution* 61(12): 2811-2821.

Perlman, W. R., Webster, M. J., Kleinman, J. E., & Weickert, C. S. (2004). Reduced Glucocorticoid and Estrogen Receptor Alpha Messenger Ribonucleic Acid Levels in the Amygdala of Patients with Major Mental Illness. *Biological Psychiatry* 56(11): 844-852.

Pickup, G. J. (2006). Theory of Mind and its Relation to Schizotypy. *Cognitive Neuropsychiatry* 11(2): 117-192.

Pickup, G. J., & Frith, C. D. (2001). Theory of Mind Impairments in Schizophrenia: Symptomatology, Severity and Specificity. *Psychological Medicine* 31(2): 207-220.

Pinkham, A. E., Hopfinger, J. B., Pephrey, K. A., Piven, J., & Penn, D. L. (2008). Neural Bases for Impaired Social Cognition in Schizophrenia and Autism Spectrum Disorders. *Schizophrenia Research* 99: 164-175.

Preston, S. D., & de Waal, F. B. M. (2002). Empathy: Its Ultimate and Proximate Bases. *Behavioral and Brain Sciences* 25(01): 1-20.

Regard, M. (1981). *Cognitive Rigidity and Flexibility: A Neuropsychological Study.* Unpublished PhD Dissertation. University of Victoria, British Columbia.

Reitan, R. M. (1955). The Relation of the Trail Making Test to Organic Brain Damage. *Journal of Consultant Psychology* 19(5): 393-394.

Rochat, P., & Striano, T. (2002). Who's in the Mirror? Self–Other Discrimination in Specular Images by Four- and Nine-Month-Old Infants. *Child Development* 73(1): 35-46.

Röhricht, F., Priebe, S. (2004). Ego-Pathology and Common Symptom Factors in Schizophrenia. *Journal of Abnormal Psychology* 192: 444-446.

Rössler, W., & Lackus, B. (1986). Cognitive Disorders in Schizophrenics Viewed From the Attribution Theory. *European Archives of Psychiatry and Neurological Sciences* 235(6): 382-387.

Russell, L., & Hill, E. J. (2001). Action-Monitoring and Intention Reporting in Children with Autism. *Journal of Child Psychology and Psychiatry* 42(3): 317-328.

Russell, T. A., Reynaud, E., Herba, C., Morris, R. W., & Corcoran, R. (2006). Do you See What I See? Interpretations of Intentional Movement in Schizophrenia. *Schizophrenia Research* 81(1): 101-111.

Russell, T. A., Rubia, K., Bullmore, E. T., Soni, W., Suckling, J., Brammer, M. J., et al. (2000). Exploring the Social Brain in Schizophrenia: Left Prefrontal Underactivation During Mental State Attribution. *American Journal of Psychiatry* 157(12): 2040-2042.

Sass, H., Wittchen, H.U., Zaudig, M. (2003). Diagnostisches und Statistisches Manual Psychischer Störungen. Göttingen: Hogrefe.

Satpute, A. B., & Lieberman, M. D. (2006). Integrating Automatic and Controlled Processes into Neurocognitive Models of Social Cognition. *Brain Research* 1079(1): 86-97.

Scharfetter, C. (1981). Ego-pathology: The concept and its empirical evaluation. *Psychological Medicine* 11(273-80).

Scharfetter, C. (1995). The ego/ self experience of schizophrenics. *Schweiz Arch Neurol Neurochir Psychiatr* 146: 200-206.

Schneider, K. (1959). Clinical Psychopathology New York: Grune and Stratton.

Schneider, K. (1974). Primary and Secondary Symptoms in Schizophrenia. In M. Hirsch (Ed.), *Themes and Variations in European Psychology*. Bristol: John Wright.

Schnell, K., Heekeren, K., Schnitker, R., Daumann, J., Weber, J., Heßelmann, V., et al. (2007). An fMRI approach to particularize the frontoparietal network for visuomotor action monitoring: Detection of incongruence between test subjects' actions and resulting perceptions. *NeuroImage* 34(1): 332-341.

Sebanz, N., Knoblich, G. n., Stumpf, L., & Prinz, W. (2005). Far from action-blind: Representation of others' actions in individuals with Autism. *Cognitive Neuropsychology* 22(3): 433 - 454.

Sernyak, M. J., Leslie, D., Rosenheck, R. (2005). Predictors of antipsychotic medication change. *The Journal of Behavioral Health Services and Research* 32(1): 85-94.

Shamay-Tsoory, S. G., Shur, S., Barcai-Goodman, L., Medlovich, S., Harari, H., & Levkovitz, Y. (2007). Dissociation of cognitive from affective components of theory of mind in schizophrenia. *Psychiatry Research* 149(1-3): 11-23.

Shur, S., Shamay-Tsoory, S. G., & Levkovitz, Y. (2008). Integration of emotional and cognitive aspects of theory of mind in schizophrenia and its relation to prefrontal neurocognitive performance. *Cognitive Neuropsychiatry* 13(6): 472 - 490.

Singh, M. M., & Kay, S. R. (1987). Is the Positive-Negative Distinction in Schizophrenia Valid? *The British Journal of Psychiatry : The Journal of Mental Science* 150: 879-880.

Soderstrom, N. C., Davalos, D. B., & Vázquez, S. M. (2011). Metacognition and Depressive Realism: Evidence for the Level-of-Depression Account. *Cognitive Neuropsychiatry*.

Spence, S. A., Brooks, D. J., Hirsch, S. R., Liddle, P. F., Meehan, J., & Grasby, P. M. (1997). A PET Study of Voluntary Movement in Schizophrenic Patients Experiencing Passivity Phenomena (Delusions of Alien Control). *Brain* 120(11): 1997-2011.

Sprong, M., Schothorst, P., Vos, E., Hox, J., & Van Engeland, H. (2007). Theory of Mind in Schizophrenia: Meta-Analysis. *The British Journal of Psychiatry* 191: 5-13.

Stieglitz, R. D., & Fähndrich, E. (1988). Interrater Study for the AMDP System. *Pharmacopsychiatry* 21(6): 451-452.

Stone, V.E. & Gerrans, P. (2006). Does the normal brain have a theory of mind? *Trends in Cognitive Sciences* 10: 3-4.

Synofzik, M., Vosgerau, G., & Newen, A. (2008). Beyond the Comparator Model: A Multifactorial Two-Step Account of Agency. *Consciousness and Cognition* 17(1): 219-239.

Tager-Flusberg, H., & Sullivan, K. (2000). A Componential View of Theory of Mind: Evidence from Williams Syndrome. *Cognition* 76(1): 59-90.

Tomasello, M., Call, J., Hare, B. (2003). Chimpanzees versus Humans: it's Not that Simple. *Trends in Cognitive Sciences* 11: 153-157.

Trillenberg, P., Heide, W., Junghanns, K., Blankenburg, M., Arolt, V., & Kompf, D. (1998). Target Anticipation and Impairment of Smooth Pursuit Eye Movements in Schizophrenia. *Experimental Brain Research* 120(3): 316-324.

Trimble, M. R. (1990). First-Rank Symptoms of Schneider: a New Perspective? *British Journal of Psychiatry* 156: 195-200.

Van den Bos, E., & Jeannerod, M. (2002). Sense of Body and Sense of Action Both Contribute to Self - Recognition. *Cognition* 85(2): 177-187.

Van Holst, E. & Mittelstaedt, H. (1950). Das Reafferenzprinzip. *Naturwissenschaften* 37: 464-476.

Van Hooren, S., Versmissen, D., Janssen, I., Myin-Germeys, I., Campo, J., Mengelers, R., et al. (2008). Social Cognition and Neurocognition As Independent Domains in Psychosis. *Schizophrenia Research* 103(1-3): 257-265.

Vetter, P., Köller, O. (1996). Clinical and Psychosocial Variables in Different Diagnostic Groups: Their Interrelationships and Value as Predictors of Course and Outcome During a 14-Year Follow-Up. *Psychopathology* 29(3): 159-168.

Vogeley, K., & Fink, G. R. (2003). Neural Correlates of the First-Person-Perspective. *Trends in Cognitive Science* 7(1): 38-42.

Waters, F. A. V., & Badcock, J. C. (2008). First-Rank Symptoms in Schizophrenia: Reexamining Mechanisms of Self-Recognition. *Schizophrenia Bulletin*: 1-8.

Waters, F. A. V., Badcock, J.C., Dragovic, M., Jablensky, A. (2009). Neuropsychological Functioning in Schizophrenia Patients with First-Rank (Passivity) Symptoms. *Psychopathology* 42: 47-58.

Waters, F., & Jablensky, A. (2009). Time Discrimination Deficits in Schizophrenia Patients With First-Rank (Passivity) Symptoms. *Psychiatry Research* 167(1-2): 12-20.

Waters, F., Rock, D., Dragovic, M & Jablensky, A. (2011). Social Dysmetria in First-Episode Psychosis Patients. *Acta Psychiatrica Scandinacia* 123: 475-484.

Wechsler, D. (1997). Wechsler Adult Intelligence Scale III San Antonio: The Psychological Corporation.

Weiner, B. (1994). Motivationspsychologie. Weinheim: Beltz.

Williams, L. M., Loughland, C. M., Gordon, E., & Davidson, D. (1999). Visual Scanpaths in Schizophrenia: Is there a Deficit in Face Recognition? *Schizophrenia Research* 40(3): 189-199.

Wolpert, D.M. & Kawato, M. (1998). Multiple paired forward and inverse models for motor control. *Neural networks* 11: 1317-1329.

Woods, S. W. (2003). Chlorpromazine Equivalent Doses for the Newer Atypical Antipsychotics. *Journal of Clinical Psychiatry* 64(6): 663-667.

Zitterl, W., Urban, C., Linzmayer, L., Aigner, M., Demal, U., Semler, B., et al. (2001). Memory Deficits in Patients with DSM-IV Obsessive-Compulsive Disorder. *Psychopathology* 34(3): 113-117.

Ziv, I., Leiser, D., & Levine, J. (2011). Social Cognition in Schizophrenia: Cognitive and Affective Factors. *Cognitive Neuropsychiatry* 16(1): 71-91.